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MEDICAL AND SURGICAL REPORT OF BELLEVUE AND ALLIED HOSPITALS IN THE CITY OF NEW YORK

VOLUME II

1905-1906

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MEDICAL AND SURGICAL REPORT

OF

Bellevue and Allied Hospitals

IN

THE CITY OF NEW YORK.

VOLUME II.

1905–1906.

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ON THE IMPOSSIBILITY OF DIFFERENTIATING SO-CALLED "PARATYPHOID" FROM TYPHOID FEVER EXCEPT BY A BACTERIOLOGICAL EXAMINATION OF THE BLOOD.*

By Warren Coleman, M.D.,

Visiting Physician, Bellevue Hospital.

If cases of infection by intermediate members of the typhoid-colon group of bacilli, when pursuing a typhoidal course, cannot be distinguished from typhoid fever without the aid of laboratory methods, the introduction of the term "paratyphoid fever" is clearly unjustifiable. I shall endeavor to prove that so-called paratyphoid and typhoid fever are identical clinically and that they can be differentiated only by the recovery of the bacillus concerned from the blood.

Schottmüller introduced the term paratyphoid fever in 1901. In 1902, in a paper entitled "Types of Infection Produced in Man by Intermediate Members of the Typhoid-Colon Group of Bacilli," [1] I took the ground "that paratyphoid infections do not constitute a clinical entity; that there is as great diversity among the different types of typhoid fever, as between typhoid fever and paratyphoid infections; that the idea of the specificity of typhoid fever should be abandoned, and that the scope of its etiology should be broadened to include *Bacillus alkaligenes fæcalis, Bacillus typhosus, Bacillus paratyphosus* and certain members of the paracolon group." At the time this paper was written only about fifty cases of paratyphoid infection had been published. As the number of cases has increased and the subject has become better known, I have not only seen no reason to modify the above view, but have become more thoroughly convinced of its correctness.

A brief review of the bacilli constituting the typhoid-colon group will enable me to explain my conception of the subject more clearly. I shall consider only the bacilli pathogenic for

* Read at a meeting of the Section on Medicine, New York Academy of Medicine, April 18, 1905.

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man. Petruschky's *Bacillus alkaligenes fæcalis,* which lies just without the group on the typhoid side, must be included, because it has produced typhoidal symptoms. The other members of the group are *Bacillus typhosus,* the paratyphoid bacilli which are not identical either in biological or serum reactions, the paracolon bacilli, which likewise differ among themselves, *Bacillus enteritidis,* consisting of different strains, *Bacillus psittacosis* and *Bacillus coli communis.*

All of these bacilli have produced diseases characterized by typhoidal symptoms, with the possible exception of *Bacillus coli communis.* I grant at once that infections by these different organisms are distinct diseases *from the bacteriologist's standpoint.* But bacteriological are not necessarily clinical entities. Pneumonia and infective endocarditis are examples. Further, if clinical distinctions are to be made on the basis of biological differences in organisms, we must be consistent and give a name to each subvariety of infection. It needs but a moment's reflection to see where this policy would lead us in the typhoid-colon infections.

The pathological anatomy of the cases of infection by the intermediates which have come to autopsy has differed from that of typhoid fever. Peyer's patches have escaped, though four of six cases showed intestinal ulcers. What the ultimate significance of these facts will be, I think no one can tell. But since the number of cases studied is so small, it will clearly be wiser not to draw conclusions from them.

The Clinical Diagnosis of Typhoid Fever and Paratyphoid Infections: Twenty-one-Day Cases.—A positive clinical diagnosis of even a typical case of typhoid fever is beset with difficulty and is generally made after the fact. No case is typical, until the disease has finished its course. More than once has general miliary tuberculosis been mistaken for typhoid fever. (I do not, of course, refer to the probable diagnosis of the usual case. We are dealing with *exact,* not probable, diagnosis.)

Some of the reported cases of paratyphoid infection have been described as typical attacks of typhoid fever. Some have been mild, some severe. Among them they have presented all the symptoms and many of the complications of typhoid fever. There has been no symptom of typhoid fever constantly absent. There has been no symptom constantly present that we may not get in typhoid fever. All that the most ardent advocates of the use of

the term " paratyphoid fever " claim for it as a distinct disease is, that the cases are milder, that they are of shorter duration (I shall consider the cases of less than twenty-one days' duration later), that the mortality is lower, and that some complications, *e. g.*, myositis and purulent arthritis, are more frequent. But it must be borne in mind that these conclusions have been drawn from less than two hundred cases and that the majority of the cases reported as infections by an intermediate have not been subjected to the only final diagnostic criterion—the recovery of the bacillus from the blood. But even admitting that all the cases reported as such were infections by intermediates, I claim that one would find as many statistical discrepancies among an equal number of cases of typhoid fever taken at random.

My personal experience is limited to one case. But that case passed for a typical, mild case of typhoid fever, until Buxton found that the bacillus we had isolated from the blood, gave the cultural reactions of paratyphoid bacilli.

Short-Duration Cases.—If the *final* diagnosis of a typical case of typhoid fever is difficult, what shall be said of the diagnosis of the irregular cases? Witness the former elaborate discussions on " simple continued fever." I have no hesitation in saying that I believe the majority of these cases to have been caused by some member of the typhoid-colon group of bacilli. The time has even come when we must take into consideration the possibility of the occurrence of primary systemic infection by the colon bacillus itself. The literature upon the subject is slowly accumulating. Buxton and I have had one case which did not give the typhoid serum-reaction, and from the patient's blood we isolated *Bacillus coli communis* in pure culture.

The argument that the cases of so-called " paratyphoid fever " have been of shorter duration than typhoid will not serve to distinguish them clinically. During the past summer I have had ample proof of this at Bellevue Hospital. I do not recall having seen in any one season so many cases of short duration, and otherwise irregular, typhoid. The diagnosis in some of them was placed beyond dispute by the recovery of typical typhoid bacilli from their blood. One lasted ten days, another fourteen, and their symptoms corresponded perfectly with those of some of the reported cases of " paratyphoid fever." These cases show, then, that short duration is not distinctive of infections by the intermediates.

Serum Reaction.—By way of differentiation, certain authors have attributed much diagnostic significance to absence of the serum reaction against *Bacillus typhosus*. Even positive diagnoses of paratyphoid infections have been made upon such negative evidence. But a negative serum reaction in a case of suspected infection by some member of the typhoid-colon group means one thing only—that the agglutinins have not developed, or at least not in sufficient quantity to give a reaction. Variability in the time of appearance of the reaction is well known. I have seen it delayed to the seventy-first day of an irregular case.

Even a positive reaction with a paratyphoid bacillus at higher dilution than with *Bacillus typhosus* does not prove that this bacillus is the cause of the infection. Grünbaum and Jolly [2] have recently (January) published a paper on the inconclusive nature of the results of the agglutination reaction in cases of typhoid fever. They tested the reactions of the blood of positive typhoid cases (proven by bacteriological examinations of the blood) against paratyphoid bacilli and obtained agglutination in 70 per cent. of the cases examined. In some cases the paratyphoid bacilli were agglutinated at higher dilution than typhoid bacilli. In 55 per cent. of the cases a colon bacillus reacted with the sera, and *Bacillus enteritidis* (Gärtner) in 22 per cent.

This only confirms what Durham demonstrated some years ago that the reactions of the typhoid-colon group are special, but not specific. The various members of the group give a group reaction, that is, any immune serum may react with several or more members of the group at low dilutions (frequently at 1 : 10 to 1 : 40, more rarely at 1 : 100).

It is, therefore, necessary to exercise caution in drawing conclusions even from positive reactions. If a serum reacts at 1 : 1000 with a paratyphoid bacillus and at 1 : 100 or less with *Bacillus typhosus*, the case is probably, but not certainly, one of paratyphoid infection. However, a group reaction at 1 : 20 is sufficient for practical purposes. Prophylaxis and treatment are the same in any typhoidal case of infection by any member of the group. And it cannot be too strongly urged that the diagnosis of paratyphoid infections concerns the bacteriologist more than the practising physician.

A successful bacteriological examination of the blood, with proper identification of the bacillus recovered, becomes then the only final criterion of diagnosis in the differentiation of infections

by various members of the typhoid-colon group of bacilli. (This statement, of course, does not refer to cases of infection by Gärtner's *Bacillus enteritidis,* except when they take the typhoid form).

In further substantiation of my position I would call attention to the fact that the term "paratyphoid fever" has been discredited in Germany, Belgium and Russia.

Conclusions.—(1) That paratyphoid infections cannot be distinguished from typhoid fever except by the recovery from the blood of the bacillus concerned and its proper identification. (2) That the present state of our knowledge makes it advisable to consider typhoid fever clinically as a disease which may be caused by several members of the typhoid-colon group of bacilli. (3) That the term "paratyphoid fever" is not only unnecessary but misleading.

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THE PRACTICAL MANAGEMENT OF TYPHOID FEVER.*

By Charles E. Nammack, M.D.,

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Gentlemen—I will omit the customary introductory rhetorical apology for the triteness of the subject of this paper. The subject was your choice, and no apology is necessary, for typhoid fever, like the poor, is always with us, and general practitioners, like the members of the Bronx Society, are more interested in its practical management than in the morphological distinctions which may be extracted from the typhoid intestine for painstaking cultivation in a laboratory. Valuable as the laboratory study of this disease during the past ten years has been, there is some truth in the shrewd intimation of a recent writer that if the same time were spent in studying the patient which is now spent in studying his blood and his secretions and excretions, we might be able to save some of the 50,000 lives lost in this country every year, to say nothing of the 250,000 who recover with systems racked by the disease. If from 1821 to 1859 the mortality in the Massachusetts General Hospital was 12.75 per cent., while from 1859 to 1899 it was 15.5 per cent.,[1] it cannot be said that we are managing this disease any better than were our predecessors, who were ignorant of the bacillus of Eberth, and of the Widal reaction, and of the advantages of a differential leucocyte count. Lest I be misunderstood, however, let me distinctly disavow any intention to underrate the beneficent results of this laboratory knowledge. If Pittsburg can afford to allow her drinking water to remain loaded with typhoid bacilli that her citizens may furnish a death rate of from 124 to 144 per 100,000, while Dresden, in Germany, a city of about the same size, is anxious to remove the bacilli and reduce the death rate to 4 per 100,000,[2] that demonstra-

* Read before the Medical Society of the Borough of The Bronx, February 14, 1906.

Reprinted from the Medical Record.

tion alone justifies all the time and study given to the bacillus. It is only my purpose to deplore the fact that, although prophylaxis has made mighty strides by help of the biological knowledge of the disease, treatment of the developed cases has not kept pace with prevention.

What is the reason for this? Are we doing too much, or too little? Or are we doing the wrong things? One of the first things expected from us in private practice is a prescription at the first visit. My personal practice is to write one calling for calomel and soda in a fair-sized dose, to be followed by a saline in six hours if necessary. I prefer one good dose of calomel to the repeated administration of fractional doses, especially for children. My next duty is to write out directions regarding diet, treatment of the discharges, and the bed linen. Everything should be done for the patient, and not by him. A bedpan is the first thing to buy. The two essential aims in the treatment of typhoid fever are to feed the patient and to starve the disease. By feeding the patient is not meant stuffing him, and if the relatives can be convinced that manslaughter is not premeditated by me, I would prefer to give the patient nothing but plenty of water for the first few days. The water may be cold or warm, flavored or unflavored, colored or acidulated. Patients like to have an irrigating bottle with tube and shut-off near the bed, from which they can help themselves when the constant attendance of a nurse is not procurable. Emptying the bowels thoroughly at the outset seems to me to furnish the best condition for arresting the growth of the bacillus, although I am quite well aware that anything like intestinal disinfection during the progress of this disease is a hopeless task. The symptomatic treatment of typhoid by intestinal antisepsis excited a great deal of interest ten years ago, owing to the sturdy claims of the late Dr. Woodbridge. This treatment was tried by me at that time in the wards of Bellevue and the Gouverneur Hospitals[3] without beneficial results. Similar measures have since been advocated by others, especially recently by William Ewart,[4] who uses liquid paraffin as a non-irritating mechanical laxative both for lubrication and for cleansing, with vegetable charcoal to absorb the toxins, and castor oil daily to secure at least one action of the bowels. Ewart is particular to state, however, that sound principles rather than therapeutic formulæ should guide each case.

After the bowels have been thoroughly moved I direct the use of milk, eggs, and water, as the essential foods during the

febrile period. Fruit juices and fruit jellies and a daily cup of strained vegetable soup or broth, flavored with beef or bacon, will supply the patient with the necessary mineral salts and satisfy his longing for something besides milk and albumen water. In cases where milk disagrees, or is not digested, whey may be substituted and is preferred by some writers.[4] In other cases, buttermilk, koumiss, or peptonized milk can be taken by patients. Ice cream makes a diverting change. Alcohol may or may not be required, according to the state of the heart and the toxemia. The average case will not need it until later in the disease. Consommé, either hot or frozen, clam juice, oyster soup, thin barley gruels, the commercial malt foods or peptonoids, clarified honey, cream, and the freshly expressed juice of broiled beefsteak furnish agreeable variants. A cup of hot coffee, well diluted with milk, taken early in the morning is unobjectionable. Natural sleep should not be disturbed for food, but where a patient is stuporous he should be thoroughly aroused before food is given, to diminish the danger of inhalation pneumonia. In spite of the fact that solid food is advised by many during the febrile period and that 1,000 cases of mixed and liberal feeding, with a mortality of only 7.7 per cent., have been reported by one observer,[5] my own practice is based upon the conviction that the diet should be liquid, with the additions cited, until the evening temperature has been normal for a week. I believe that the measures of diet herein enumerated will provide ample nourishment, will leave the least possible amount of unabsorbed residue to irritate the upper intestine, and will afford abundant variety and support to the patient during the febrile period.

The indications for the use of alcohol are: First, weak heart action as shown by small, feeble, irregular pulse, with feeble cardiac impulse and faint first sound; second, profound toxemia, as shown by delirium, tremor, carphologia, or subsultus tendinum; third, respiratory complications, as bronchitis, bronchopneumonia, croupous pneumonia, or pleurisy; fourth, previous habitual use of alcohol; fifth, persons of feeble constitution and those past middle life. A contraindication to its use is acute nephritis, or chronic nephritis with an acute exacerbation due to the typhoid infection.

In private practice we are expected to order drugs. The most useful prescription, in my opinion, is a combination of hydrochloric acid and liquid pepsin given after the food. To this may be added, when indicated, strychnine. Strychnine will be needed under about the same circumstances as alcohol. But there are

some cases in which the typhoid toxins affect the nervous system in a manner similar to strychnine. These cases may be recognized by marked exaggeration of the knee jerk and a tendency to opisthotonos and to trismus. In them, strychnine should not be given.

Ten years ago,[3] I expressed the hope that the serum diagnosis of typhoid fever would soon be followed by successful serum therapy. That hope is yet to be realized. At the present day the most successful agent in the management of typhoid fever is water. The use of water, inside and outside, is not new. Since the publication of Ernest Brand of Stettin, in 1861, advocating systematic cold bathing, the method of tubbing in typhoid fever represents the most marked advance in the management of this disease. The details of this procedure are familiar to you all. Its good effects are by no means limited to the reduction of temperature. In fact, if a case of suspected typhoid fever during the first week shows a drop of more than 2½° F., after a Brand bath, it would be well to revise your diagnosis and look for some other disease. The other beneficial results of the tub bath are shown in the nervous system by lessening of the toxic features; in the circulation by reduction of the pulse and rise of the blood pressure; in the respiratory organs by diminution of the initial bronchitis and passive congestion; in the skin by lessened liability to bedsores; in the kidneys by augmented excretion of urine and of toxins, and in the mortality by a reduction which, when first reported, seemed incredible. It is the only routine measure of treatment for which I leave a standing order in hospital practice. But the labor and expense cannot always be borne by families. It has been estimated that the average cost of each Brand bath in private practice is two and a half dollars, and when a serious case requires several baths in a day for several successive days, the cost might be prohibitive. We are then often obliged to accept a substitute for it. Cold sponging is an excellent alternative and will suffice for a case of moderate severity, but cases with very pronounced nervous symptoms will require the cold pack with sprinkling or rubbing with ice over the pack. Other practical home modifications of the bath treatment may be devised, or will be found described in special treatises, but none of these can fully take the place of the tub bath.

Special symptoms and complications in typhoid fever require the utmost watchfulness on the part of the physician. Headache in the first week is better treated by quietude, darkness, and cold

locally, than by coal tar derivatives. Sleeplessness may be persistent and exhausting and demand bromide or chloral. Opium has a bad influence upon digestion and the secretions in the early days of typhoid fever. Somnolence, stupor, and delirium must be treated by stimulants and external antipyretics, and a delirious typhoid patient must not be left alone for an instant. Tremor is an indication for the use of alcohol and strychnine. Dry tongue and sordes will not be prominent if mouth disinfection has been carefully attended to. Vomiting, when occurring early, means either profound infection or extreme gastric irritability. Later in the disease it may be of uremic origin. Constipation, after the first week, should be treated by enemas cautiously given. Diarrhea may require change of diet, especially if much milk be taken, and full doses of bismuth combined with small doses of Dover's powder. If tympanites develop, in spite of the empty bowel treatment which has been advised at the outset, the old-fashioned turpentine stupe, with perhaps turpentine enemas, will be useful. F. F. Caiger, in the Bradshaw lecture for 1904,[6] advises oil of cinnamon (in doses of 2½ to 5 minims, every 2 to 4 hours, in gelatin capsules or emulsion) for this unpleasant symptom, as well as for its influence as an intestinal antiseptic and its soporific effect in many patients. A rectal tube may be of service if the gas is in the lower bowel.

For intestinal hemorrhage occurring in typhoid my standing order used to be immobility, ice, and opium. Latterly I have discarded the opium, because it masks the signs of perforation—which is apt to occur in hemorrhagic cases. These signs must be recognized promptly if the patient is to have any chance for his life. Ergotine, hypodermically, or adrenalin, now takes the place of opium, but it is extremely doubtful if any drug of the vasoconstrictor class has any real effect in checking intestinal hemorrhage. Collapse is best met with the subcutaneous injection of sterilized salt solution, about a pint being given in from 2 to 4 places. External heat, absolute quiet, absolute deprivation of fluids by the mouth, posture, and hypodermic injections of stimulants will all be required in addition; but we must carefully steer between overstimulation on the one hand and fatal syncope on the other. There is no longer any doubt about the necessity for surgical operation in cases of perforation, nor any wisdom in selecting cases for operation. Every patient should have this chance of rescue from certain death, and the only contraindication to operation is a moribund condition. Early diagnosis and early op-

eration mean the saving of one-third of these cases, and the weight of opinion and experience is now against waiting until the second twelve hours. The symptoms of perforation should be constantly in mind in every case of typhoid, and should be promptly recognized before peritonitis supervenes to cloud the picture and to render surgical help futile. A peculiar change in the facial expression associated with a feeble running pulse and shivering often precedes the development of the characteristic pain. This pain is of sudden severe onset and increasing intensity, localized at first in the right iliac fossa but soon becoming general. Tenderness and rigidity, followed by symptoms of collapse, and later by abdominal distention, make up a suggestive picture, but it is doubtful if there is a single distinctive symptom, although muscular rigidity *newly arisen,* with coincident increase in the pulse rate, may furnish the keynote of diagnosis.

Conditions which may simulate perforation are pleurisy, pneumonia, cholecystitis, perforation or rupture of gallbladder, acute gastro-intestinal indigestion, iliac thrombosis, appendicitis, sloughing of a mesenteric gland, peritonitis, renal calculus, distended urinary bladder, rupture of an acute splenic abscess, and abdominal myositis. In cases of doubt it is better to operate, as typhoid patients stand exploratory laparotomy very well. Cases of peritonitis not due to perforation will not be injured by laparotomy. It must never be forgotten that perforation often occurs without the fulminant symptoms supposed to be characteristic.

Besides intestinal perforation, one other spectre stands by the side of every typhoid patient, and that is cardiac asthenia, due to poisoning by the typhoid toxins. The treatment of this condition consists in its prevention by elimination and support during the early stages. Once developed, its immediate outlook is bad, and its remote prognosis very uncertain.

The chest complications of typhoid fever may be overlooked if careful watch of the respirations, as well as of the pulse, is not kept up. In a case attended by a trained nurse, a bedsore should not be accepted either as a symptom or as a complication of typhoid fever. It is neither. It is a blunder. A moderate range of temperature requires no treatment. High temperature does not demand either antipyretic drugs or guaiacol externally. Water will control it more easily and safely. The nervous symptoms of typhoid will likewise respond to ice and water much more comfortably than to depressants, hypnotics, or opiates. Cholecystitis occurs in typhoid fever much oftener than it is recog-

nized, and is believed by John H. Musser and others to be the condition existing in many cases of so-called relapse. If its symptoms are very severe and progressive, operation may be necessary. A true relapse, however, is a fresh attack of typhoid fever and must not be confounded with the many other causes of post-typhoid sepsis. Besides cholecystitis, there may be inflammation of the urinary bladder, or of connective tissue, and although all these are due to the typhoid bacillus, they are not typhoid fever. These septic temperatures will not respond to baths as will typhoid temperatures, and here let me repeat the value of this differentiating information. Some of these will soon be made plain by the evidences of thrombosis of vessels. Others probably depend upon a new streptococcal infection coming on after the typhoid infection, and some of this latter group will do best if they are taken out of bed and fed. Bacilluria causing typhoid cystitis is much more manageable since the advent of urotropin—one of the new drugs that has come to stay.

According to Sir William Broadbent, the convalescence from typhoid fever should last at least a year, and in many cases longer. This period is necessary because the heart suffers from the toxins of infectious disease to such an extent that a certain amount of fatty degeneration is almost the rule. And not only the heart, but the muscular coat of the arteries is likewise similarly affected. The influence of typhoid fever in producing permanent circulatory changes was not appreciated in this country until emphasized by W. G. Thayer.[7] Many cases of arteriosclerosis in early life may be traced to a previous typhoid infection. The convalescence of the average patient may be harder to manage than was the febrile period, but the wise physician will issue just as careful orders regarding diet, exercise, and emotional disturbance, and will decline responsibility unless they are followed. In spite of all care, there is a long line of possible medical and surgical complications and sequelæ of typhoid fever, which subsequently may be attributed by the patient to leniency or lack of firmness on the part of the medical adviser.

The limits of this paper preclude any discussion of the prevention of typhoid fever. It seems strange that a disease which, if every man did his duty, would not exist at all, is still allowed to go on, and that the people of the United States pay an annual tribute of ninety million dollars to its existence.[8] The medical profession can strive to remove municipal ignorance by education,

and to show corporate greed by exposure, but this unnecessary sacrifice of life and money will go on until the public conscience is thoroughly aroused.

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TWO CASES OF RELAPSING FEVER; WITH NOTES ON THE OCCURRENCE OF THIS DISEASE THROUGHOUT THE WORLD AT THE PRESENT DAY.

By Robert J. Carlisle,
Assistant Visiting Physician, Bellevue Hospital.

INTRODUCTION.

The first of these two cases, exceptional not only in its occurrence but in some of its clinical features also, shows that other cases occur from time to time which are mistaken because of the mildness of the symptoms or insufficient care in examining the blood.

There are no other cases on record in this country up to the present time in which *Sp. Obermeieri* has been found in the blood.

The origin of the first case has not been directly traced, but the existence of cases in tropical America, hitherto unsuspected, seems to indicate its source and makes its appearance here less surprising.

The second case was the result of accidental inoculation.

CLINICAL HISTORY.

Case I.—C. N., 39 years of age, an Englishman and by occupation a ship's steward, walked into Bellevue Hospital at 9 a. m. on August 15, 1905, and was admitted to the Third Medical Division.

Previous History—He arrived in New York on August 11, 1905, on a steamer of the Galveston Line plying between New York, Galveston, Tex., and Key West, Fla. He had served three trips on this steamer as steward, being five days at sea and three days in Galveston each trip. He knew of no cases of illness on board nor in Galveston nor in his boarding house in New York. Prior to this he had spent six months in Santo Domingo and Porto Rico, had made several voyages to South America, and the West Indies during the last five years, and had not been sick during this time. Most of his sea life previously had been spent between England, China, and the East Indies. Twelve years ago (1893), while in the coasting trade out of Bombay and Calcutta, had malaria (?) fever. The attack lasted six weeks. He never had had typhoid fever nor syphilis.

Present Illness—On August 13, 36 hours previous to admission, he had a distinct chill lasting half an hour, and following the chill some

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sweating, continuing probably an hour and a half. He had severe frontal headache and did not sleep well that night. On the next day, though he had no chill, he suffered from chilliness, headache and thirst. That night he felt weak and could not sleep and concluded to go to the hospital the next day. From the 12th he had had three thin stools each day; no vomiting; no epistaxis; no cough.

On admission his rectal temperature was 102.2° F. and pulse 96, regular and of good force, the chief complaints being severe frontal headache, general aching pains, and some stiffness of the legs. On his face, neck, and upper part of trunk there was an erythematous blush; a few very small pink macules which disappeared on pressure and returned were seen on the trunk. Mucous membranes appeared normal; lymph nodes in neck and groins were just palpable. Tongue was moist, broad, slightly tremulous, with slight brown coating. The spleen was palpable and somewhat tender on pressure. There was little tympanites and some slight tenderness over the descending colon. A few sibilant râles were heard over both lungs. There were no other objective symptoms nor abnormal signs. He was a spare man, fairly well developed and nourished, and weighed 130 to 135 lbs. He was placed on a milk diet at first. Medication throughout his run of fever was placebo.

During the afternoon of the day of admission patient had a slight chill followed by a fall of temperature to 98.5° at 1 A. M. of the 16th. The leucocyte count was 7,800, Widal reaction negative, 1 to 10. (The differential blood counts are given in the table below.)

August 16. Temperature at 3 P. M., 101.6°, pulse 92, respirations 24. No malaria parasites seen on careful search. Urine: 30 oz., sp. gr. 1014, faint trace of albumin, few small hyaline casts; no diazo-reaction. The temperature declined steadily after 3 P. M. to 96.4° at 5 A. M. on 17th, the pulse receding to 52. Patient fairly comfortable with hot bottles and blankets. Has had two or three loose stools during the past two days.

On August 17, 18, and 19 patient was comfortable, no headache, no abdominal tenderness or distension; stools formed. Spots have disappeared. Spleen is palpable. The pulse reached 48, temperature subnormal.

August 20 (seventh day). At 3 P. M. temperature 98.5° F., pulse 56. The second paroxysm now began, the fever rising steadily until by noon of 21st it reached its highest point, 104.4° F., pulse 96, respirations 24. Bowels constipated, headache, shiverings, and general discomfort. Total urine 44 oz.

Examinations for malaria parasites were unsuccessful. Temperature declined sharply (97° F. by 9 P. M.), with some sweating. Pulse 52, respirations 18.

August 24, 9 P. M. (three days after last paroxysm). The temperature, which had been subnormal for three days, rose to 100.2° F., pulse 88, declined quickly to 97.2° F., pulse 68, and by midnight of 25th had reached 96.5° F., pulse 56. Meanwhile the spleen remained enlarged, bowels constipated. There was no eruption. Three days later, 27th, a similar rise to 99.6° with acceleration of pulse was noted, and disturbed sleep.

With these two exceptions the temperature range was subnormal

almost continuously (a few times reaching 98.5 F.) for nine days until 6 P. M. of August 30. Patient was out of bed and on regular diet. During this interval the patient's temperature was taken every three hours and daily searches were earnestly made for the malaria hemamœba by every member of the staff, all without success. It was this persistent scrutiny, however, that was finally rewarded by the discovery by the house physician, Dr. Heitlinger, of spirochetes in a stained smear made just after noon of August 31—temperature 102.4°. He found six in one smear (1-12 immers., oc. II.).

This, the third distinct paroxysm, began at about 6 A. M. August 30, and continued for 36 hours. At the beginning he asked for an extra blanket, but he had no distinct chill at any time during the pyrexia. He had severe headache and said he felt "mean all over;" he says he can tell when the attack is coming on because he dreams the night before and is wakeful.

The spleen is tender and more easily palpated. During the decline of temperature there was a return of the looseness of bowels; five stools. Blood taken during night of 31st and forenoon of September 1 contained about six spirochetes to each smear. On September 2, 24 hours after the crisis, one spirochete was found after careful scrutiny of four smears. Highest temperature of this paroxysm was 103° F. Crisis occurred in early hours of September 1, and by 9 A. M. the temperature was 98.5°.

The pulse ran higher during this paroxysm than in first two (112 to 118). Promptly after the crisis the patient felt well, diarrhea ceased, splenic tenderness subsided but the spleen remained palpable.

On and after September 2, the patient was up and about every day during the rest of his stay in hospital (till November 9) except at the time of the fourth and last period of pyrexia (September 9 to September 11). A constant watch was kept every three hours on his temperature until October 6. Weight September 2, 130 lbs.

There is nothing notable of this last intermission except that the spleen remained palpable and patient's weight increased to 134 lbs. His appetite improved; he felt well. He had been placed on a tonic of Fe et Potas. Tart. and Port wine.

September 9. During night of September 9 and 10 patient was restless and could not sleep; had severe headache; felt cold but had no chill; temperature at 5 A. M., 97.6° F., pulse 80, respirations 28. Fever began and by midnight of 10th reached 103° F., pulse from 112 to 120. Face flushed, skin hot and dry, erythematous blush appeared as in the first paroxysm. Spleen enlarged and tender; bowels loose. A remission of 2.5° F. occurred during night with some lessening of pulse rate, but at 9 A. M. with a temperature of 100.4° F., pulse was still 108.

The temperature then rose steadily during the afternoon, reaching the highest point of the fever at 7 P. M., 104.5° F., the patient meanwhile being greatly distressed with frontal headache and muscular pains, and had a pretty severe chill. Pulse 124. Crisis was suddenly ushered in at 7 P. M. by sweating and fall in temperature (7.5° in 10 hours) and coincidently marked amelioration of all the symptoms. Increased looseness of bowels was noted. Weight 131 lbs., a loss of three pounds during the

paroxysm. September 13, complained of considerable pain in right shoulder. No objective signs.

Spirochetes.

Sept. 10	2 P. M.	Temp. 101°	1	in 13 smears.		
	10	" 102.8	3	" 2	"	
	12	" 103	7	" 2	"	
Sept. 12	11 A. M.	" 98	None	" 4	"	
Sept. 15	11	" 98.8	"	" 4	"	

From this time on convalescence was uneventful. The temperature ranged between 97° F. in the early morning and 98.5° in the evening until the 25th, after that it ran about normal until patient's discharge on November 9, except that several times it rose to 99.6° F. as on the 19th and 20th (nine days after last paroxysm) and on the 28th, when the patient reported as being restless and uneasy. Nothing untoward developed though the spleen could be felt. By October 1, he had pretty well recovered from his anemia, the differential count was nearly normal. No spirochetes could be found. The spleen could not be felt. On discharge patient weighed 152 lbs.*

Each attack of fever that was observed was characterized by prodromic wakefulness and restlessness; chilliness, rapid, though not abrupt, rise of temperature beginning in the forenoon; decided increase of pulse-rate compared with the interval; severe headache; moderate enlargement of spleen; high percentage of large lymphocytes (and in the first and fourth paroxysms by an erythematous rash) and ended in a crisis, the rapid fall in temperature to subnormal occurring during night hours with some diarrhea, sweating, and marked fall in pulse-rate. No jaundice was observed nor gastric symptom at any time. The features of the intervals were subnormal temperature, slow pulse, and rapid return to a feeling of well-being. The patient was not very sick at any time.

Spirochetes—The organisms found were to all appearances identical with *Sp. Obermeieri*. The accompanying photographs (1500 diam.) furnish the best description, all three of which were taken from the same microscopic slide. The organisms were not very numerous. They were first seen in the stained specimen (Wright's stain). They varied in form and size; they appeared in increasing numbers during the access of fever and disappeared rapidly after the crisis. Only once were any seen during the interval, as is noted above. This was about 36 hours after the crisis. Three days later the unsuccessful inoculation was made.

* I take this opportunity of thanking Drs. Heitlinger, Rimer, and Satchwell, of the house staff for their unremitting care and attention given in this case.

No cases developed in the ward or hospital as a result of the presence of this case which was treated in general ward, no special means being taken to prevent infection.

CASE I.—URINE EXAMINATIONS

	August.					
	16	17	19	21	29	31
Stage of Disease.	1st Parox'm.	Day After Crisis.	1 Day Before Parox'm.	2nd Parax'm.	1 Day Before Parox'm.	3rd Parox'm
C.c. in 24 hours	887	1,124	1,124	1,301	887	1,124
Sp. Gr.	1,014	....	....	....	1,010	....
Urea 24 hours, grammes	...	....	....	....	7.77	....
Reaction	Ac.	....	....	....	Ac.	....
Albumin	Faint Tr.	....	....	....	None.	....
Sugar	None.	....	....	....	None.	....
Diazo	None.	....	....	....	....	....
Microscope	A few small hyaline casts.	....	....	....	A few small hyaline casts.	....
Diet	Milk.	Milk.	Milk.	Milk.	Unrestricted Diet.	

	September.					October
	1	7	9	11	12	2
Stage of Disease.	Day After Crisis.	3 Days Before Parox'm.	1 Day Before Parox'm.	4th Parox'm.	Day After Crisis.	21 Days After Crisis.
C.c. in 24 hours	1,360	2,070	2,839	1,715	2,129	1,479
Sp. Gr.	....	1,015	1,014	....	1,012	1,016
Urea 24 hours, grammes	....	22.68	27.99	....	20.99	9.72
Reaction	....	Ac.	Ac.	....	Alk.	Neut.
Albumin	....	None.	None.	....	None.	None.
Sugar	....	None.	None.	....	None.	None.
Diazo	....	...	....	....	....	....
Microscope	....	Neg.	Neg.	...	Cryst. and Amorph. Phosph.	Neg.
Diet	Unrestricted Diet.					

On September 5, four days after a paroxysm and three days after the last spirochete was found, a portion of 7 c.c. of blood

drawn from the arm of this patient was injected directly subcutaneously into a monkey (*Macacus rhesus*). There was no reaction. On September 10, during the access of the last paroxysm, 5 c.c. was withdrawn from the patient and 3 c.c. injected subcutaneously.* It was during the manipulations incident to these experiments that the second case developed. The clinical account of this I present in the words of the patient himself.

Case II.—The patient was one of the writers of the article that follows. Infection probably took place through being bitten by one of the monkeys, during the latter's paroxysm. After the fracas incidental to having their temperature taken, the monkeys frequently bled from the gums, and it is reasonable to suppose that spirochete blood was inoculated with the bite in this way. This may have occurred on one of several occasions, and therefore the exact period of incubation cannot be made out with certainty.

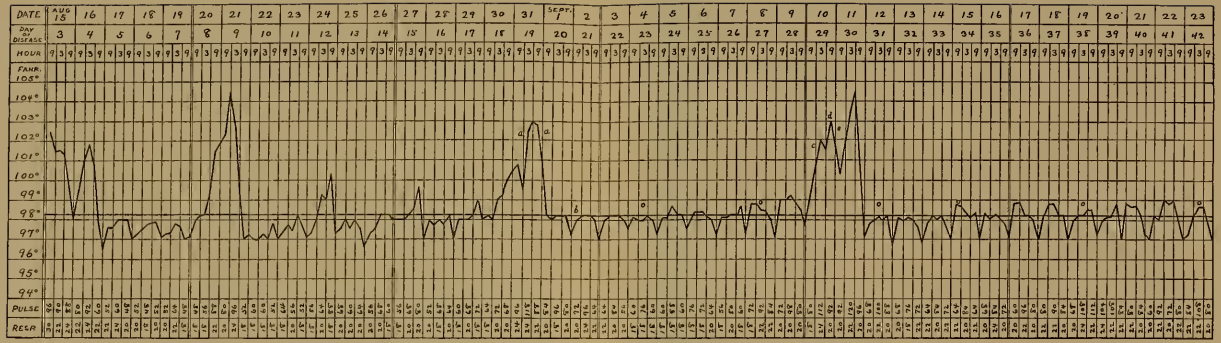
Family and previous history have no bearing upon the case, and may be omitted.

On the afternoon of October 8, the patient was suddenly taken ill with chilly sensations, severe frontal headache, shooting pains in the extremities, and moderate general prostration. This increased during the night, and the following afternoon he took to his bed. Appetite was completely lost from the beginning. There was no nausea or vomiting, and no icterus; bowels constipated.

The temperature began to rise about 12 hours after the onset of the subjective symptoms, reaching 103° on the afternoon of the second day. It then fell to 100° and remained comparatively low until the fourth day, when it reached 105°; this was followed after a few hours by a critical defervescence; marked by a drop to normal within 12 hours. The first paroxysm, therefore, lasted four days. Following the crisis there was an apyrexial period lasting for seven days, during which the temperature was persistently subnormal, ranging from 95° to 98°. During the interval there was an absence of all subjective symptoms. The patient was out of bed on the second day, regained strength and appetite rapidly, and was completely recovered before the onset of his first relapse.

On October 18, seven days after the first crisis, there was a recurrence of the subjective symptoms marking the onset of the first attack. After a prodromal period of about 12 hours, the temperature again rose, reaching 102° on the afternoon of October 19. During the ensuing four days there was remittent pyrexia. On the morning of October 21, the temperature fell to normal, to rise again to 101.8° during the day. The following morning it fell to 95°, then rapidly rose, and at 8 P. M. reached its highest point, 105.4°, to fall by crisis to subnormal during the night. These remissions in the temperature were attended by a transient release from all feelings of illness and discomfort.

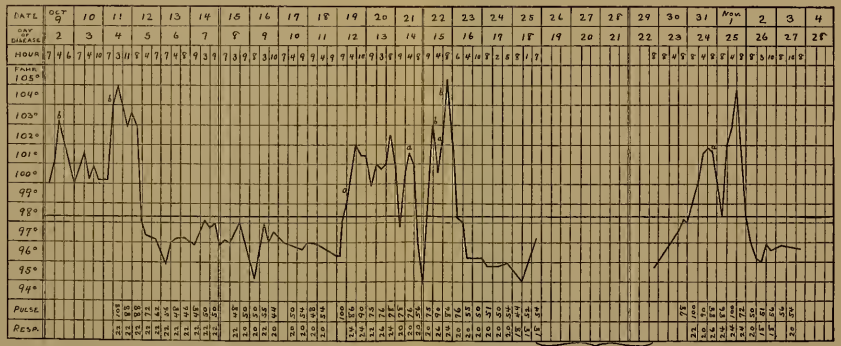
* The further results of this and other animal inoculations are reported by Dr. Norris and his assistants, Drs. Pappenheimer and Flournoy, in the article that follows in this number of the Journal.



*RELAPSING FEVER CASE, I., C. N., BELLEVUE HOSPITAL, AUGUST 15, 1905.

No malaria organisms in daily search.

a, 6 spirochetes in 1 smear. *b*, 1 " " 4 smears. *c*, 1 spirochetes in 13 smears. *d*, 3 " " 2 " *e*, 7 spirochetes in 2 smears. *o*, No " " 4 "



No records taken.

RELAPSING FEVER CASE II., OCTOBER 8, 1905.

a, Spirochetes present, not numerous. *b*, Spirochetes present, fairly numerous. *o*, Spirochetes absent.

The second paroxysm, therefore, lasted for four days. There succeeded a second apyretic interval of eight days.

At the end of this period there was a recurrence of the usual symptoms, accompanied by a febrile paroxysm lasting two days. On the morning of the second day, there was a remission to normal, followed by an abrupt rise to 105°, and an equally abrupt crisis during the following night.

The third paroxysm, therefore, lasted for but two days. The temperature remained subnormal for several days, then gradually rose to normal. Convalescence after the third attack was rapid and progressive. Only for a few hours, on the 12th day after the final crisis, was there a recurrence of the sense of nervousness and malaise marking the onset of the previous attacks. There was no febrile reaction, however, and this may have been an incidental condition unrelated to the illness.

The course of the disease as outlined above is fairly typical of the relapsing fever caused by the *Sp. Obermeieri*. The duration of the febrile paroxysms is somewhat shorter than the average; yet great variations in the length of the pyrexial and apyrexial periods are known to occur.

Certain individual symptoms may be commented upon. The pulse-rate throughout the febrile period remained relatively slow, as compared with the temperature. It rarely exceeded 100; ranged usually about 80, and on one occasion, with a temperature of 105.4°, was only 76. A marked bradycardia accompanied the subnormal temperature between the paroxysms, the pulse-rate ranging from 40 to 50.

Chilly sensations were frequent throughout the greater portion of the fever; during the first relapse, with a sudden rise of temperature, there was a severe, shaking chill, which lasted about six hours. This was the only real chill that occurred.

Darting pains in the limbs, and especially in the inguinal regions, were present with each attack, and at times were quite distressing. A dull pain in the back, and in the splenic region was also felt at times.

The sudden falls of temperature were attended by very profuse sweating, usually coming on during the night. These drenching sweats also persisted for several nights after each crisis.

The spleen, examined daily, was palpable only once (October 20, first relapse), and did not appear to be much enlarged. Slight edema about the eyelids was seen in the early mornings, but disappeared after a few hours.

The urine showed a trace of albumin and a few granular and hyaline casts. The excretion of urea was increased. The diazo-reaction was negative.

The blood naturally showed the most interesting changes. Spirochetes were present during each febrile period, disappearing after the crisis. They were never numerous, however, and during the last paroxysm were found but once out of four examinations, and in very small numbers; this, too, in spite of the high temperature and severe constitutional symptoms.

The leucocytes counted on October 9, the second day of the illness, were 14,400. Unfortunately no subsequent counts were made.

A marked and striking relative, and probably also an absolute, increase in the number of large mononuclear cells, or megacaryocytes, occurred with each attack. This is well shown by the following differential counts, made at various times during the illness. Transitional forms and degenerated polymorphonuclears were also in excess during the febrile period.

Aside from this the blood showed little of interest. Polychromatophilia of any degre or granular degeneration was not present.

Convalescence after the second relapse was exceedingly rapid, and save for slight loss of weight and weakness after severe exertion, the patient was in normal health within a week after his final crisis.

Treatment was entirely symptomatic, and apparently in no way influenced the course of the illness.

RELAPSING FEVER.

Blood Records.*

CASE I. 300 WHITE CELLS COUNTED.

Day of Disease.	3	19	21	23	27	30	38	50
Stage of Disease	1st Par'x'm 3d Day.	3d Par'x'm 1st Day.	3d Inter. 2d Day.	3d Inter. 4th Day.	3d Inter. 8th Day.	4th Parox'm 2d Day.	Last Apyrex'l Period	Last Apyrex'l Period.
Time and Temperature.....	1–3 P.M. 101.5°	1–3 P.M. 102.5°	1–3 P.M. 98.2°	1–3 P.M. 98°	1–3 P.M. 98.8°	1–3 P.M. 103°	1–3 P.M. 98.4°	1–3 P.M. 98.6°
No. of reds.....	....	....	....	4,100,000	....	3,400,000	3,600,000	4,480,000
No. of whites...	7,800	....	....	5,600	....	5,700	7,600	7,600
Ratio.....	....	....	....	1:732	....	1:596	1:473	1:589
Hb% (Tallqvist)	....	....	....	80	....	70	70	90 +
Polymorph.....	73%	50%	54.2%	55.6%	59.6%	62.1%	....	64%
Small lymph..	25	17.3	10.8	7.2	2.3	5.5	....	33
Large lymph..		27.5	30.6	31	27.9	24.5	....	
Mononuc. and transition...	1	5	4.2	5	5.7	5.5	....	1.5
Eosinophile.....	0.5	0.1	....	1	3.5	1	....	0.5
Mast cells.....	0.5	0.1	0.1	0.1	1	1	....	1

* All the blood-counts of Case I. were made by Dr. Satchwell of the interne staff; those of Case II. by the patient himself.

CASE II. 100 WHITE CELLS COUNTED.

Day of Disease.	2	6	12	14	15	24	25	25
Stage of Disease	1st Parox'm 2d Day.	1st Inter. 1st Day.	2d Parox'm 1st Day.	2d Parox'm 3d Day.	2d Parox'm 4th Day.	3d Parox'm 1st Day.	3d Parox'm 2d Day.	3d Parox'm 2d Day.
Time and Temperature.....	P. M. 103°	P. M. 97°	12 M. 99°	P. M. 101°	P. M. 103°	11 P.M. 100.8°	1 P. M. 101.8°	7 P. M. 104.8°
No. of reds.....	....	....	....	....	....	....	....	....
No. of whites...	14,400	....	....	....	....	....	....	....
Ratio.....	....	....	....	....	....	....	....	....
Hb %(Tallqvist)	....	....	....	....	....	....	....	....
Polymorph	....	40%	71%	61%	68%	67%	71%	79%
Small lymph..	Incre'd	6	6	....	3	3	4	1
Large lymph..		9	16	3	1	9	1	2
Mononuc. and transition...	....	43	4	35	32	21	24	17
Eosinophile.....	....	2	4	1	1	....	0.	1
Mast cells	....	....	....	....	....	....	....	....

SUMMARY.

	Case I.	Case II.
Duration of paroxysms	3, 1½, 1½, 2 days	4, 4, 2 days
Duration of intermissions	3, 9, 9 days	7, 8 days
Highest rectal temperature recorded	105.5° in last relapse	105.4° in first relapse
Lowest rectal temperature recorded	96.4° in first crisis	94.8° in second crisis
Highest pulse-rate recorded	124 in last relapse	110 in second relapse
Lowest pulse-rate recorded	48 in first crisis	44 in first and second crises
Greatest fall in temperature at crisis	7.5° in 10 hours	8.8° in 17 hours
Greatest fall in pulse rate at crisis	124—68=56 at last crisis	110—47=63 in last crisis

[1] Marked granular degeneration of the red cells.

On a cursory view of the temperature and other features of Case I., there is nothing suggestive of relapsing fever to anyone not well acquainted with this disease, and even then he might miss the diagnosis, for so good an observer as H. Vandyke Carter, speaking of the first cases he saw in 1885-86, says, "without special attention it is always possible that genuine spirillum fever may be confounded under 'Remittents.'" But when a spirochete is found in the blood which bears a distinct relation to the paroxysms and is identical in its morphology to that described by Obermeier, and when a second case develops by infection from this one which strongly resembles the typical descriptions of relapsing fever, one can hardly avoid the conclusion that the disease in question is really one of true relapsing fever.

The paroxysms in both cases resemble those of relapsing fever in the abrupt onset with chill or chilliness; the frequent recurrence of this symptom just before the crisis at the acme of the attack; the moist skin and sweating during the febrile periods; and in Case II. drenching sweats during, and for some time subsequent to, the crisis; the moist tongue—this is mentioned by Murchison as being the rule throughout the attack, and Flint says it is somewhat distinctive of this disease. The severe headache, muscular and leg pains are very characteristic, and arthritic pains without inflammatory or other joint symptoms not infrequently occur. The moderate enlargement of the spleen and the splenic tenderness are other points of resemblance. And lastly, the crisis occurring during the night time far more frequently than during the day time (Lebert, Carter, Strümpell, and others). The intermissions were marked by extraordinarily low temperature and pulse records, especially in Case II., and this for days at a time.

Variations occur in this disease, as Strümpell says, as well as in any other disease, and it is this fact that Case I. emphasizes.

SUMMARY OF CERTAIN CLINICAL FEATURES OF RELAPSING FEVER.

The Duration of the Initial Paroxysm—Murchison says in rare instances it does not exceed three or four days; Flint says, exceptionally, it may be only two days; Carter says it may end at any time after the fourth or fifth day.

The Duration of the Intermission—The longest Murchison says was 12 days; it may not exceed two or three days, and it may extend to 12 days or more (Flint); sometimes only four or five days, rarely two weeks or so (Lebert, who believed relaps-

ing fever and bilious typhoid were not identical); from 6 to 12 days and may extend to 15 days (Sandwith).

Duration of Relapses—Many instances could be given of variation in length from a few hours up to, and even longer than, the primary fever.

Number of Relapses—Murchison says five may occur "making in all six paroxysms"; Flint never saw more than one; Lebert says: "I had not formerly believed in a fourth relapse until during the last epidemic several cases presented it undeniably"; and, finally, Strümpell says the more accurately and persistently we take the temperature during convalescence, the oftener do we find slight rises of temperature occurring at intervals late in the history of the case (see chart of Case I. and note the remark in history of convalescence of Case II.).

Remissions in the Temperature—Often there are oscillations to a considerable degree (Strümpell). In Carter's 270 cases in 1876-78 pseudo-crises were remarkable in both invasion and relapse, and in his cases in 1885-86 he says the perturbations of temperatures were marked. Other instances might be cited occurring both before and after Obermeier's discovery.

It should be noted that we can hardly compare the charts of these two cases to those in the textbooks in this respect, as the former represent three or four-hour intervals, while the latter generally show only morning and evening variations.

Pulse-rate—Usually high in the paroxysms, it may vary from 90 to 110, according to Murchison, but he also says there is less correspondence between the pulse and the temperature in the relapse than in the first paroxysm, instancing a temperature of 106° F. with a pulse of 90 (see Case II. of this report—temperature 105.4°, pulse 76).

Nausea and vomiting are among the most common symptoms, sufficiently so, as Flint says, to be somewhat distinctive, especially when the disease is contrasted with typhus and typhoid fevers. In our two cases they did not occur in the first case, but in the second there was some nausea shortly after the crisis. Nor did the great enlargement of the spleen, reported by some, exist in our cases. The spleen as a rule, however, seems to be but moderately enlarged.

Our two cases differed as regards the condition of the bowels: Case I. had diarrhea frequently, and in this case sweating was much less prominent than in Case II.

These two cases then show considerable similarity to the clinical history of relapsing fever as presented by the older writers and the variations which do occur are within the limits of variation recognized by them. Care should be taken that a sporadic instance of disease be not made to conform with earlier descriptions, and, on the other hand, the case in question should not be classified as a new discovery unless it fails in some important particular to conform with the previously recognized type, or until some other way is known of identifying the spirochete than by its morphological characteristics.

HISTORICAL AND GEOGRAPHICAL.

That relapsing fever existed was made plain by the British observers of the eighteenth and the early part of the nineteenth centuries, especially by Henderson (1843), Wm. Jenner (1849), and their contemporaries, and was so well differentiated from typhus and typhoid fevers that Clymer was able to recognize it in this country in 1844, and Griesinger was able to report that most of the cases of bilious typhoid in Egypt in 1851 were in reality relapsing fever. Later, when in Europe it was seen in epidemic form, it was supposed to be a disease indigenous to the British Isles.

Obermeier's demonstration, which gave the requisite criterion whereby the disease might be known in sporadic cases and variant types, was soon confirmed by Carter in India, who was the first Englishman to find the organism and to produce the disease in animals (monkeys).

So that there are two important periods in the natural history of relapsing fever. One may be said to begin with Henderson of Edinburgh, in 1843, and the other period may be said to begin when Obermeier in 1873 announced the discovery, which he made in 1868, of the organism which bears his name.

British Isles—The years 1868-71 mark the last appearance of this disease in the United Kingdom, at least in epidemic dissemination. For 15 years before that it had not appeared. Moore, writing in 1891, says he had never seen a case, though he had seen service in two hospitals in Dublin for nearly twenty years, one of them being a large epidemic-fever hospital.

Whereas before Great Britain and Ireland had been an epidemic center, the disease has ceased to prevail there now for nearly forty years.

Russia—Eastern Europe has had many visitations, especially Russia. It is said to have occurred first in 1833 at Odessa and in 1840 at Moscow (Hirsch, Dehio). A great epidemic raged in St. Petersburg in 1863-65; in Livonia, one of the Baltic provinces, in 1865 and again in 1883-84; Finland, 1865; Siberia, 1866, and Poland, 1868 (Hirsch), and smaller outbreaks over Russia in 1863-68 (Dehio). Clemow, quoting Reitlinger, of St. Petersburg, says it has reached as far north as Archangel, that in 1874 St. Petersburg was the great center, and that yearly reports came from Vologda, a northeastern province which had been formerly free. It existed in St. Petersburg in 1879-82, 1885-86, and 1890-91; Novgorod, 1898; it was rife in Warsaw in 1879 and 1883, in Moscow in 1882, and again in 1894-95 and 1898.

The observations of Gabritschewsky, of Moscow, on the development of immunizing substances (1896) may be mentioned here, Löwenthal's serum-diagnosis (Moscow, 1898), and Sawtschenko and Melkich, also on immunity, in the province of Kasan on the upper Volga in 1900.

In 1893-95 the largest number of cases were reported from near the Volga in the southeast, and the smallest from the Baltic (Clemow, Dehio). Odessa has, from first to last, often been an epidemic center. Moschutkovsky of Odessa, 1876-79, produced the disease in healthy man by the inoculation of infective blood. Tictin, writing of the cases occurring in Odessa about 1889 or 1890, says relapsing fever occurs about once in every 15 or 20 years there.

The United States consul-general's office reports the total number of deaths in Russia from relapsing fever in the year 1901, as given by the Medical Department, as 2,466, out of a total of over 700,000 deaths from infectious diseases (including therein 250,000 from diarrhea and dysentery).

In 1905 the disease continued to figure in the mortality records of St. Petersburg, Moscow and Odessa. The *United States Consular Reports* state for the week ending May 6, 4 deaths and 19 cases in St. Petersburg; for the three weeks ending May 13, 7 deaths and 126 cases in Moscow, and for the fortnight ending May 13, 1 death and 81 cases in Odessa, and yet, as the *U. S. Public Health and Marine Hospital Service Reports* point out, the malady has not been introduced into this country notwithstanding the large immigration.

Germany has been infected principally in the eastern part and probably from Russia (Hirsch). Upper Silesia, according to

Murchison, showed cases in 1848, concurrently with typhus. The most important visitations, however, were those that took place from 1868-73 through northeast and south Germany and which gave rise to the brilliant work of Otto Obermeier in Berlin. Appearing first in Silesia in 1867, the disease spread the next year to Berlin, Stettin, Posen, Königsberg, and other cities. Again in 1878-79 other small epidemics occurred in Berlin, Dantzig, Magdeburg, Halle, Dresden, Breslau, and Würzburg, Heidelberg, and Giessen—1879-80. Since then Germany has been free from epidemics.

In 1895, however, there appeared an imported sporadic case, which is especially noteworthy because of the possible bearing it has on the mode of infection in view of the recent work on human tick-fever in Africa. This case was discovered in Hamburg among the emigrants by Dr. de la Camp, who says it is the first case that had occurred in the Emigrant Bureau Hospital.

De la Camp's Case.—The patient, a Persian on his way to the United States, entered hospital July 15, 1895, on the fourth day of his illness. Seventeen days previously he had started from Ooroomeyeh [a city of about 25,000 people, in the northwestern part of Persia, 64 miles southwest of Tabreez. The interest of this lies in the fact that the tick *Argas persicus*, which is said to cause disease in man, is principally found in Persia, in the neighborhood of Miana, a place 80 miles southeast of Tabreez]. The journey was made by horses to Tiflis; rail to Batoom, and thence to Hamburg by rail (steamer Batoom to Odessa).

On admission the temperature was 40.4° C., pulse 100; three paroxyms occurred of eight, six, and three days' length, respectively, intermissions of five, and thirteen days' length. Fever in first relapse developed gradually during three days, and during the relapses was quite remittent. Pulse during first and second fever periods was below 100, except twice 120 was noted; during third pyrexial periods 120 to 160. Crises were acute with marked sweats—greatest fall in second, 41° to 34° C., pulse 125 to 64. Each paroxysm was accompanied by headache, and muscle and joint pains, constipation, slight jaundice, enlarged and tender spleen, and a trace of albumin in the urine near the time of crisis, these symptoms disappearing during the intervals when the temperature became subnormal for a day or two. Spirochetes were discovered one hour after the rise of temperature in third paroxysm (12 to cover glass)—not found after crisis. Leucocytosis marked, but less during relapse and absent during intervals. Hb. 55 per cent. The case is reported in full.

Austria—Relapsing fever, recognized first as bilious typhoid at Cracow in 1847, occurs nowadays especially in Bosnia and Herzegovinia. In 1889-90, in the latter province, an epidemic is reported upon by Karlinski at Stolac. This observer made some investigations on the transmission of this disease by bugs. The

disease was epidemic in these parts in 1903. Hödlmoser, of Sarajevo, Bosnia, has done some work on serum diagnosis.

In the *Balkans* the disease is not frequently referred to, and according to Clemow in recent years has been absent from Constantinople. The well-known description by Hippocrates, in which he seems to allude to relapsing fever in the island of Thasos, off the coast of Salonica, may be referred to here, and it may be noted that he gives four symptoms occurring at the crisis, namely, epistaxis, encuresis, dysentery, and sweating, that are of good omen.

The disease is said by many to be found occasionally in Greece, the Ionian Islands, and the Ægean Archipelago—Crete also, and Cyprus—in which places it goes principally under the name of bilious typhoid, which Griesinger pointed out and later Engel proved to be often really relapsing fever.

Asia Minor, Palestine, and Syria—Cases are sometimes discovered in this part of the world. Tictin, at Odessa, in 1889 or 1890, saw a case in a sailor who had just arrived from a voyage which included a stay at Jaffa, where he seems to think the infection originated. Jaffa lies 31 miles northwest of Jerusalem. As the voyage was of some length, however, the infection might have originated on board ship. The latest record is by J. C. Cropper, at Jerusalem, July 15, 1905, who found a spirochete in the blood of a boy 15 years old who had never been out of the country. The boy was reported sick with a "fever of a quartan type." His temperature was 101° F. and he was not very ill. The organisms were not very numerous ($\frac{1}{12}$ immers.). G. C. Low, of London School of Tropical Medicine, who examined the slides, reported that the spirochete was probably *Sp. Obermeieri*. Cropper says it is the second case he has seen.

Clemow says relapsing fever was never prevalent in *Transcaucasia*. Isolated cases occurred in 1889-90 in the City of Tiflis. Quoting Pantiukhoff, who wrote in Tiflis in 1898 on the "Statistics of Caucasian Pathology," he says that sporadic cases occurred along the railroad during the years 1894-98.

Of Mesopotamia and Persia, little or nothing is recorded. The disease is not uncommon in Western Siberia in the Government of Tobolsk.

India—The disease has long been recognized in all parts of India and constantly recurs. The most important epidemic, from a historic point of view, was that which occurred in Bombay and

its neighborhood in 1876-78. It was then that Carter made his important announcement confirming the presence of *Sp. Obermeieri,* and stating that "spirillum fever" is identical with relapsing or famine fever or typhus recurrens; that it can be readily communicated to the monkey (*Macacus radiatus*) and is "unequivocally manifested by both marked general symptoms and abundance of the attendant blood parasite," and that "post-mortem appearances are equally concordant."

In a letter to the *Lancet* in March, 1880, he made the important statement that he had induced spirillum fever in monkeys by inoculating with infected blood taken during the prefebrile incubation period. Seven years after the close of this epidemic Carter saw five undoubted cases of relapsing fever with Obermeier organisms in the blood, that is from September, 1885, to April, 1886, and by January, 1888, was able to report six cases more, proving, as he says, the continued presence in Bombay of this typical disease, and that notwithstanding the general prosperous condition of the local population.

In 1899 relapsing fever was discovered by Waters in the Common Prison at Bombay, the cases having been first reported as plague. The diagnosis was made by blood examination. Cuthbert Christy reports finding in September, 1900, while "at Ahmednegar, perhaps the healthiest and nicest station in the Bombay Presidency," in cases of an epidemic disease in a near-by village, *Sp. Obermeieri.* In this same village plague had occurred the previous year. The disease, he adds, is endemic in certain quarters of Bombay. He made some observations at this time on infection by bedbugs, of which there are plenty there.

There can be no doubt that spirillum fever, to use Carter's term, is continually present in Bombay. Clemow (1903) says the disease, relapsing fever, was "severely epidemic during the last three years." A. Powell, of the British army, expresses the opinion that there must have been "tens of thousands" of cases in Bombay in 1901-2, and adds that it is strange it is not imported into Europe, the journey to London requiring but 14 days.

Amongst the hill tribes of the Himalayas it has long been known. According to Murchison it is said to have been recognized in 1852 in the valley of Peshawur, and again Hirsch shows that it was probably introduced into the Punjaub by the muleteers returned from service in Abyssinia in 1866. In the Kumaon Hill in 1898, Leon Rogers reports relapsing fever, and finds the *Sp.*

Obermeieri in one of a group of cases all having similar symptoms and reported as mild plague by the natives. Also G. Browse reports finding this organism in a case simulating malaria occurring in March, 1904, at Nowshera in the Punjaub, in a well-situated and healthy regimental station. The patient was a cavalryman who had not been away from his station for a year. The case, he says, was exactly like cases diagnosticated as malaria in the Punjaub.

L. A. Walker found the parasite in some cases of a recurrent fever epidemic in the eastern portion of the Mardan district on the right bank of the Indus in 1904.

As to *Farther India and Malay Peninsula* the only mention I have found is that of Dr. James Kirk of Singapore, in a paper read before the Malay Branch of the British Medical Association, Session 1903-4, analyzing 150 cases of local fever. He says: "I have not yet diagnosed relapsing fever here."

East Indies—A case of relapsing fever is reported in May, 1901, from Sumatra, by J. C. Graham at Deli, in which the spirochete was found. The patient was an immigrant Chinese coolie from Swatau. He says it is the first case ever reported in Sumatra. The symptoms were high fever, severe headache, pains in the legs and joints, tenderness in both hypochondriac regions, slight icterus. No malaria parasites were present, specimen teemed with "*Sp. Obermeieri.*" Crisis on sixth day with fall to 35.6°.

China—In the north, according to report, various foci existed in times past. In 1864-65 this disease was epidemic concurrently with typhus fever, and Tien Tsin suffered greatly in 1877. It was reported in the province of Shan Tung as very prevalent in 1889 (Neal—no blood verification).

In the south, the most common form of fever at Ting-Chau-fu is relapsing fever, to quote Clemow again.

From the province of Kwang Tung on the southeast coast spirochetic infection in sporadic instances is recently recorded from two places. One of these cases, namely Swatau, was mentioned above. The others are by L. G. Hill, of the Church Missionary Society Hospital, at Pak-hoi on the gulf of Tong King. In July, 1903, in making routine blood examinations of all cases, he discovered in a woman 65 years old "*Sp. Obermeieri.*" She had had a fever period of 11 days, an intermission of three days, and fever again for four days according to report, when admitted.

A crisis occurred the same night. An interval of nine days followed, then a period of three days' fever, a one-day interval, and a one-day relapse. The organisms were many and active when first found. During the first two days of the first interval that was observed, the organisms continued in diminishing numbers and then disappeared till next paroxysm, when they were again plentiful, but not so active. Though the case was treated in the general ward, no other case developed.

On March 7, 1904, a Chinese coolie, aged 33 years who had been in the employ of a European for several years and who lived in the coolie quarters, was admitted with a temperature of 101°, having been ill several days. Myriads of parasites were found. No malaria organisms—excess of leucocytes. Crisis occurred two days later, the temperature registered 99.8° and no spirochetes were found. March 10, submormal temperature and no organisms.

Hill says the disease had not been previously met with at Pakhoi nor at Hong Kong.

In January, 1905, Koch of Hong Kong met with three cases among Chinese emigrants en route from Chin-wan-tao to South Africa. The periods of these cases were as follows:

Case I.—Two paroxysms of seven and four days with five days' intermission. Case II., fever 12 days, no "remission;" Case III., fever 12 days—sudden death after three days of apyrexia. Numerous "spirilla were found and no malaria." He states he is unable accurately to determine the duration of the first paroxysms. In the fatal case "no spirilla were found in any of the organs or in the blood of the heart. The spleen was enlarged and firm."

As a sequel of these cases the office boy of the hospital fell sick of the disease and went through a typical and severe attack with fever periods of six and ten days, separated by an interval of five days.

This observer says the disease is very rare in Hong Kong.

The Public Health Reports of the United States Marine Hospital Service for June 2, 1905, say that cases occurred in various places in China in the first part of last year.

Japan—Baelz suggested in 1898 (as quoted by Clemow) that an epidemic disease which he observed in the island of Skikoku in 1881, may have been relapsing fever. Scheube (1903), who was formerly connected with the University of Kioto, does not speak of this disease as occurring in that country.*

* One or two papers were published at Tokio in 1895 on recurrent fever, printed in Japanese.

Philippines—I have been unable to obtain verification of the statement of Osler (1905) that these islands have suffered from several severe outbreaks—neither in Hirsch, Davidson, Clemow, Scheube, nor elsewhere.†

Egypt—After the clinical demonstration by Griesinger, and the microscopical proof given by Engel at Cairo in 1884, we hear little of relapsing fever in Egypt till 1904. Clemow, writing in 1903, says the disease was formerly endemic in Egypt, but that it is absent from reports throughout Africa in general.

A good clinical account of this affection as it is seen in the Delta is given by F. M. Sandwith, of Cairo, in May, 1904. Sandwith found the spirochete in cases reported as typhoid in 1884 in and about Cairo and at Zagazig, an important cotton center 75 miles northwest of Suez. It appears that the disease has been more or less continually present ever since. Eight deaths occurred in the provincial prisons in 1902. During the previous 11 years he had altogether 35 cases in his wards in Cairo. Suez, on the highway to the East, is another important center of distribution. Sandwith says the only case he ever saw in a European was at this place in 1892. In July, 1905, there is a report of five cases at Cairo imported from Suez, in which the spirochetes were found by L. Phillips. The temperature charts are given. The shortest relapse was three days; the longest interval 10 days.

Abyssinia is noted above in connection with the Punjaub.

Tripoli, Tunis, Algeria—Along the north coast of the African continent the disease has been reported recently by several of the French army surgeons.

A. Billet reported a case in an Algerian soldier, aged 30 years, in September, 1901.

The man came to the hospital at Constantine with the diagnosis of malaria. He had two paroxysms: the first lasted four days; the second one and one-half days; the interval lasted 13 days. A few "Obermeieri" were seen the first day (four or five to field) and they increased to 10 or 15 to field before crisis. There was a prompt disappearance at the crisis. In the relapse a few were found (1 in 10 or 15 fields). A leucocytosis of 26,600 when the temperature was 39° C. on third day: polymorphonuclears, 77 per cent., large mononuclears, 1 per cent., small mononuclears, 22 per cent., eosinophiles, 0.1 per cent., 100 cells counted. The reds numbered 2,511,000. Hb., 85 per cent. (Henaque). After the crisis the white cells fell to 19,000 (65 per cent. polymorphonuclears) and

† The report of Assistant Surgeon B. L. Wright, U. S. N., in the Philadelphia Medical Journal, 1901, 7, p. 301, of cases of "Cavite Fever" puts one strongly in mind of relapsing fever. Although no reference is made to this disease in the article it is possible the cases were of that nature.

in second paroxysm the count was 17,000 (74 per cent. polymorphonuclears). Other features were absence of prodromes, slightly remittent temperature, rapid pulse (120 to 125) and respirations, enlarged spleen, some icterus, dry tongue, delirium, epistaxis preceding crisis, abundant urine and profuse sweat at crisis, followed by subnormal temperature.

Billet says the disease is excessively rare in Algeria. A small epidemic occurred near Constantine in 1866. Friant and Cornet record some cases occurring in Algeria near the frontier of Tunis in December, 1903, and January, 1904.

Lafforgue reports in 1903, from the vicinity of Tunis, 20 cases in which a spirochete was found identical in form and mobility and other characteristics with *Sp. Obermeieri*. He thinks cases probably occur in Tripoli.

Morocco—It is said by both Davidson and Clemow to have appeared in this country during the periods of famine following plagues of locusts.

Grand Canary—In the *U. S. Consular Sanitary Report* for the week ending May 6, 1905, 13 cases of relapsing fever were reported in the Grand Canary (Spanish).

Gibraltar—Here may be noted the case of infection by spirochetes that was reported by Manson in 1904. An English lady, aged 30 years, who, having resided at Gibraltar for three years, returned there after a short visit to England on September 29, 1903. On October 20 she had a rigor followed by a fever (104° F.) which after profuse sweat terminated in four days. A ten-day intermission and a second paroxysm occurred, lasting three days, after five days a third, lasting also three days. Another interval of 10 days, during which time the patient returned to England, and on November 24 a fourth period of fever began. After this paroxysms occurred on December 4, 15, 29, and January 11. Each paroxysm except the last, which was very mild, was ushered in by rigor and terminated with profuse sweating. Spirochetes were found in the paroxysm of December 29, but were not looked for before, except during the subsiding fever of the previous period. They were few in number, and Manson is noncommittal as to whether it was the *Sp. Obermeieri* or not. He thinks there were too many relapses and that the spirochete (of which an illustration is shown) differs in shape from this well-known organism.

The number of paroxysms is greater than in any other case I have found record of.

Tropical Africa—In the course of the years 1904 and 1905 several clinical and three most important experimental reports were made under the various titles: relapsing fever, spirillum fever, spirillosis, and "tick-fever" from British East Africa, the Congo Free State, German East Africa, and Portuguese West Africa.

Uganda—A. R. Cook, from the missionary hospital at Mengo, at the head of Lake Victoria Nyanza, published, early in 1904, some notes of cases of relapsing fever.

Case I.—March 21, 1899. A little girl, whose blood teemed with spirilla, was admitted in extremis and died 16 hours later; no post-mortem examination. During the next four years isolated instances were seen, and in November, 1903, a somewhat widespread epidemic was noted.

Case II.—November 7, 1903; a man ill three days with vomiting and pains in the chest; no rigor; no diarrhea; collapsed condition; temperature 99.4°, pulse 140, respiration 80; conjunctivæ jaundiced; delirious. Spleen and liver not felt; very scanty urine, trace of albumin. Fresh specimen of blood showed over 30 spirilla to one field ($\frac{1}{12}$ immers., oc. III); death in four hours. Autopsy three-fourths hours later. Smears from the enlarged spleen showed few spirilla.

Case III.—November 8, 1903. A man three days sick in his second paroxysm. Rigor at the onset. No vomiting nor pains. Temperature 101.6°, rising in one hour to 104.2°; slight delirium; no jaundice. Blood showed one organism to each field. Crisis on fourth day when spirilla disappeared.

Case IV.—November 15, 1903. Small girl whose blood swarmed with spirilla. Temperature 105°; no accurate history.

Nabarro, while studying sleeping sickness in Uganda, met with a case of spirillar infection on August 1, 1903.

Hodges and Ross, also in Uganda, discovered the organism in the blood of an Indian who had been absent from India for over a year and who had never had a similar attack before. These organisms were few, sharply pointed at both ends, and were about 40 μ long and 4 μ broad. The symptoms were vomiting, back-pain, temperature 103.4°, with spleen slightly enlarged and tender. Crisis on the next day; relapse 18 days later when spirilla were again found. A monkey was inoculated. After three and one-half days fever began in the monkey, lasting three days; numerous spirilla. Every case of fever was examined (60) coming from various parts of the Protectorate, and 12 showed spirilla, comparatively few organisms in all. In the one fatal case the spirilla disappeared from the blood 24 hours before death, while the temperature was still high.

Angola—Portuguese West Africa is a province bounded on the north by the Congo State. From Benguela, a district on the coast of this province, in the early part of last year A. Y. Massey reported a case of spirillosis in a man which he met with in December, 1903. The patient, a Portuguese trader, had a temperature of 103.4° and involuntary passages, and died three days after admission. Fresh specimens of blood showed spirilla innumerable. Next, Wellman, in April, 1905, reported from Angola a case of relapsing fever with spirilla. The patient, a native 32 years old, was admitted March 3, 1904, one day sick; temperature 104.4°, pulse 92, respirations 29. No rigor or vomiting; organisms about a dozen in each field ($\frac{1}{12}$ immers., oc. II.). Crisis occurred on the third day. A few spirilla persisted for two days more. After an intermission of seven days a relapse of three and one-half days occurred during which the organisms were fewer in number.

Ile de Réunion and Mauritius—These islands off Madagascar were infected with relapsing fever in the year 1865, the disease having been introduced direct from India by coolies. It lasted for several years (Hirsch and Stephen Smith).

Uganda (Tick Fever)—Continuing the investigations of Hodges and Ross at Entebbe, Uganda, Philip H. Ross and A. D. Milne, knowing of Marchoux and Salimbeni's demonstration that spirillosis of fowls is conveyed by a tick, thought that perhaps the so-called tick-fever might be due to a spirillum. They were unable to get any reliable description of symptoms, nor good temperature records. Some cases had vomiting and some had relapses; some had splenic tenderness. The most common symptoms were severe headache, pains in back and limbs, and fever ending critically. All had spirilla sparsely scattered and difficult to find. They concluded that certain cases diagnosticated as tick-fever are due to a spirillum. Their observations were published on November 26, 1904. On the same date J. E. Dutton and J. L. Todd cabled from the Congo: "Spirilla cause human tick-fever. Naturally infected ornithodoros infect monkey."

Congo Free State—Dutton and Todd were working independently of Ross and Milne and were on the Upper Congo at Kasongo some distance west of Lake Tanganyika in November, 1904, when they both contracted this disease which later led to the death of Dutton, February 27, 1905. A report of their work was issued last November (1905) and their conclusion is that

the tick-fever in the oriental province of the Congo Free State is a relapsing fever, produced by a spirochete, probably identical with *Sp. Obermeieri,* and that this organism can be transmitted by the tick *Ornithodorus moubata.* The report contains records of 14 cases of which two cases were Europeans, Dutton and Todd themselves being the victims. There are also notes of animal experiments, and remarks on the distribution of the human tick in the Congo region, together with notes on the external anatomy of *O. moubata* by Newstead.

Dutton and Todd remark what Livingston said, that the ticks followed the Arabs, and point out that the ticks seem to have come into the Free State by two routes: from the East Coast with the Arabs, and with traders from the Portuguese territory to the south. A closely allied, if not identical, ornithodorus is found in Angola (Newstead).

Cuthbert Christy found these ticks in Uganda in 1902, and says that they are distributed in German East Africa, Angola, and in the Zambesi, and are closely allied to the *O. Savignyi* found in Egypt, Nubia, Abyssinia, and Somaliland.

Milne says that one of the chief caravan roads in German East Africa, south of Victoria Nyanza, had been closed, owing to constant sickness caused by tick-fever. Koch found the ticks widely distributed over the district (1905).

The tick *Argas persicus* is found in Persia and Beluchistan, being especially common, according to Manson, in the northern part of Persia in the neighborhood of Miana. The natives attribute a disease to its bite, which is known as the "disease of Miana." (Compare de la Camp's case noted above.) Manson quotes Schlimmer as saying that on one occasion (1858) he treated 400 soldiers who declared they had been bitten by these ticks at Miana. The symptoms resemble those of "remittent fever—extreme lassitude, disinclination to work, yawning, fever, perspiration, not accompanied by much thirst, increasing and decreasing at stated hours of the day."

United States—The first introduction of relapsing fever into this country was by Irish immigrants in 1844 at Philadelphia, when Clymer was the first to recognize it, he having 15 cases under his care. Pepper, Parry, and others also saw cases. The disease did not spread far. In 1850-51 Flint differentiated cases from among some Irish immigrants affected with typhus, who arrived at Buffalo where he was then in practice, and he also

reported 15 cases, and adds that doubtless others occurred which were mistaken for typhus. It is mentioned by Clymer that, in a report from the Russian government to the English ambassador at St. Petersburg, the disease was said to exist at New Archangel (now Sitka, Alaska) in 1858. The next and last time it appeared here was in epidemic form in New York. The first case appeared in October, 1869. The epidemic lasted through the winter and spring and in rapidly waning numbers into 1871. In Philadelphia numerous cases appeared. The disease did not spread far from these centers, however. A few cases occurred in Washington, in Maryland, New Jersey, and Connecticut, and one case was imported into Boston, as noted by Shattuck in the American translation of Strümpell's work on "Practice of Medicine."*

In September, 1874, a severe outbreak of epidemic disease was observed at Oroville, California, among Chinese laborers. Many of these cases were similar to typhus, judging from the description, but many had the characteristics of relapsing fever; crisis on the sixth day and relapse on the 14th day after a period of complete apyrexia, and are so reported by the observer, Miller.

At Worcester, Mass., in August, 1899, a sporadic case appeared in an Armenian immigrant and is recorded as relapsing fever by Ward. This diagnosis was not confirmed by blood examination, though the spirochete was looked for during the relapse. The case presents all the other characteristic features, however. Malarial organisms were absent.

South America—Baldou says it appeared in Peru in 1854 and, known as "peste des Cordillères," spread thence slowly to Chili and Bolivia (1856). Hirsch (1881) says nothing is known of its existence in Central and South America.

Cuba—Relapsing fever is next heard of in this part of the world in 1902, when Biada, of Havana, reports, under the title "recurrent fever," an important case that simulated yellow fever and by a careful examination of the blood was proved to be due to spirochetic infection.

* A case of relapsing fever entered the Presbyterian Hospital, New York, last July (1905), in the service of Dr. George A. Tuttle, who kindly permits me to mention the fact. The patient, an Armenian woman who had been in this country for a number of years, had entertained, a few days before her sickness began, some relatives newly-arrived from Armenia; they themselves were not sick, however. The patient had two paroxysms of fever, each of five days' duration separated by an interval of like length. The diagnosis was confirmed by the finding of the organism in the blood during the relapse. The case is to be reported in the Medical and Surgical Reports of the Presbyterian Hospital for 1905-6.

Biada's Case.—Spaniard (Espanol) aged 19 years, a sailor for one and one-half months on steamer from Liverpool, to the coast of "Cantabricas" (Spain?), to Cienfugos, to Havana, and then on a coasting schooner. The onset was abrupt on January 19, 1902, with chill. Temperature 40° C., pulse 116, respirations 30, pains all over, especially in legs; constipation; spleen very large. No malaria parasite was seen in the blood, but perfectly characteristic *Sp. Obermeieri* (three in one field) were discovered by Dr. Kirby Smith. The urine (550 c.c) contained some albumin on third day. The fourth day much pain in splenic region, diarrhea; fifth day, epistaxis; sixth day, black vomit, slight jaundice, crisis—fall from 103° F. to 95.4° from 6 A. M. to 6 P. M.; disappearance of spirilla. On the seventh day felt better; stools formed, urine 325 c.c., less albumin. Eighth and tenth days, urine between 1,400 and 2,200 c.c.; felt fine on 11th day; no spirilla. February 7, 13th day after the crisis, epistaxis returned, urine contained much albumin, and again, six days later, no fever appearing and no organisms, abundant albumin was noted. February 20, slight iritis. He was subsequently discharged without having any relapse of fever, but with some edema of extremities, a little albumin in the urine, and bad color to skin.

The day before the crisis (January 24) a monkey was subcutaneously inoculated with 3 c.c. of patient's blood. Spirochetes were found in the monkey's peripheral circulation 61 hours after inoculation. The animal's temperature ranged between 40° and 39° C. for two days and fell on third day coincidently with the disappearance of spirochetes.

Mexico—Some cases were noted here in April, 1905. The Surgeon-General's office of the U. S. Public Health and Marine Hospital Service writes in answer to an inquiry, that they were reported from Tuxpam, that deaths occurred from this disease during every month throughout the year, but that the weekly sanitary reports from United States Consul Lesspinasse at Tuxpam do not state the number of cases occurring, nor give other information relative to the deaths that have occurred. Tuxpam is a seaport on the gulf coast between Vera Cruz and Tampico—145 miles northwest of the former place. It has a large trade in cedar, vanilla, fustic, and coffee.

Panama—The Surgeon-General has very kindly furnished the following statement, dated February 14, 1906: "It is understood that two cases of the disease (relapsing fever) were under treatment in the Colon Hospital, Colon, Panama, during the summer (1905), the diagnosis having been verified by blood examination."

The foregoing account shows that there are cases of periodic fever occurring in many widely separated parts of the world which, in their clinical manifestations and infective character, are strikingly similar to relapsing fever, and, as regards the associated microorganism parallel, if not identical, therewith. In respect of

so-called tick-fever, Dutton and Todd, in their report, are as explicit as Carter was. They say (p. 4): "The case reports, charts, and post-mortem finding certainly demonstrate the clinical identity of the tick-fever observed by us, with the relapsing fever of the textbooks." Koch says that the relapsing fever seen by him in 1905 in German East Africa is closely similar to the European relapsing fever, the unlikeness being that the relapses are shorter and the spirochete is, as a rule, longer in the African disease.

MODE OF INFECTION.

Most, but not all, of the older writers believe that relapsing fever is a contagious disease.

Many authorities might be cited and many instances given to show that the disease is not a highly contagious one; that, on the introduction of a case into an uninfected area, the disease does not spread rapidly nor far; that from the existence of a single case in cleanly surroundings, and without special attempts at isolation, the disease is not newly developed.

None of the reasons given, tending to prove that the disease is a contagious one in the sense that typhus fever is said to be, is inconsistent with the view that it is conveyed by blood-sucking insects, such as have been proved to be the means of infection of spirochetic disease, as in so-called tick-fever in man, *Sp. Theileri* in cattle, spirillosis of fowls, etc.

Nuttall says that Flügge, in 1891, was the first to suggest the possibility of vermin serving to spread relapsing fever. Tictin, in 1897, at Odessa experimented with the ordinary bedbugs, and succeeded in causing the disease in monkeys by inoculating them with blood taken from bugs immediately after they had fed, but failed to succeed when an interval of 48 hours was allowed to elapse.

Karlinski found abundant spirochetes in bugs caught in houses in Bosnia, where relapsing-fever cases existed, and none in bugs caught in other houses free of this disease. He says that spirochetes persisted for 20 days in bugs. Dutton and Todd found living spirochetes in the stomach and malpighian tubules of ticks up to five weeks after their feed on a known infected animal, and were able to infect a monkey after one and three-fourths months. One of their conclusions is that the transmission (of the spirochete) is not mechanical, but some developmental process is carried on in the tick.

Schaudinn suggests that certain stages in the life-history of the *Sp. Obermeieri* may be formed in the blood of the patient which in relapsing fever are not recognized as parasites.

Cuthbert Christy at Ahmednegar, India, did not succeed in acquiring the disease by allowing himself to be bitten by bed-bugs caught in the bed of a patient sick with relapsing fever.

In Case I. of this report, the disease undoubtedly originated outside of New York. That spirochetic disease existed in the part of the world where this patient has spent much of his time recently has been shown—Havana, 1902; Colon, 1905, and perhaps cases at Tuxpam. No case developed in Bellevue Hospital subsequent to his stay of 89 days in hospital, and absolutely no special means were taken to prevent infection. There are several varieties of ticks to be found in the West Indies, South and Central America (*O. talaje*) and Mexico (*O. turicata*). Sambon has suggested the *O. turicata* in Mexico as a possible factor.

DIAGNOSIS.

There is great approach to unanimity shown by the various observers in the terms employed, namely, relapsing fever, recurrent fever, recurrent typhus or spirillum fever. Furthermore, a study of all these reports and our cases leads one to the conclusion that these cases cannot be differentiated one from another either clinically, or by the morphological features of the spirochete, as far as these are known, or by animal inoculation.

A thorough search of the blood during the pyrexial period, and according to Carter, even during the pseudo-crisis, will reveal the spirochete. These are not very numerous in many of the cases. They increase in number with the progress of the febrile period, and in each succeeding relapse, because of decrease in numbers, more care is required in the search. They begin to disappear just before the crisis, but may be found (in Case I. in four large smears and in one of Hill's cases at Pak-hoi and the first two cases of Dutton and Todd) a day or two after the fall. The statement of Sandwith, that the spirilla entirely disappear from 12 to 24 hours before the temperature falls at the crisis, is certainly not true for most of the cases, though Heidenreich, of St. Petersburg, quoted by Fagge, makes a somewhat similar statement. Heidenreich mentions what has been spoken of by many of the recent observers, that there are fluctuations in the number of the organisms during the same pyrexial period, and

differences in mobility, size, and shape may be noted in the same specimen of blood (Carter, Dutton and Todd, and others, and see accompanying microphotographs from the same smear of blood in Case I.). Carter and Heidenreich have found the spirochetes during the interval, just before the relapse, and in Cases 8 and 9 of Dutton and Todd the day before the rise of temperature. They were seen in the interval also in one of the native "tick-fever" cases, by Ross and Milne—the patient complaining of severe headache, but without a coincident return of fever. Sometimes when a relapse is about due, the patient complains of some subjective symptom, headache, wakefulness, restlessness, and the temperature is slightly raised (subfebrile), but no spirochetes are found. (Fagge and Cases I. and II. of this report.) The organisms have been found after death, in the blood and in the spleen (Strümpell, Dutton and Todd's Case IV. and Cook's Case II.).

The blood, besides the positive proof presented during the febrile period, shows some changes in the leucocytes which apparently vary in greater or less degree, side by side with the progress of the disease.

Many of these cases, in which the number of white cells are recorded, show a moderate leucocytosis (de la Camp, Hill, Billet) particularly distinct in the first paroxysm. Case I. of this report, and of the three instances of Dutton and Todd's report in which the note is made, Cases XIII. and XIV., do not agree with this, in the other of these three instances, the fatal case, the count was 14,200 and during the crisis 11,800.

It may be said that there is very often recorded, in the first paroxysm, an increase in the number of white cells, particularly the polymorphonuclears, which diminishes early in the interval and is less marked with each recurring paroxysm. This is not invariable, as is shown in the table given above, in which the most marked change is in the relative number of large lymphocytes and mononuclears. No distinct changes in red cells other than those indicative of secondary anemia occur.

A study of these sporadic instances of spirochetic infection suggests that the following symptoms may indicate the probability that this condition is present.

A period of fever of four or five days' length, beginning abruptly and ending abruptly, chilliness or chill at onset, recurring just before the crisis. Sometimes epistaxsis at the same periods, very severe headache, backache, and leg pains. Mod-

erate sweating and moist tongue during the fever. Moderate enlargement of spleen, with considerable tenderness on pressure. Pulse increased, perhaps rapid. Recurrence of similar paroxysms at any time from third to eighteenth day, usually between seventh and fourteenth. Relapses short, from one-half to three days as a rule, during which fever may be higher than in first paroxysm. The number of relapses may be from one to seven—perhaps more. Often a pseudo-crisis the day before the true crisis. Crisis accompanied by drenching sweats coincidently with a rapid and very great fall of temperature occurring during the night and extending over 12 or 16 hours.

The interval characterized by temperature and pulse-rate much below the normal standard; this often continuing until the day before the next paroxysm. Sweating persisting often for two days at the beginning of the interval; disappearance of splenic tenderness and quick return to feeling of well-being.

Löwenthal has proposed a serum reaction which (when the opportunity offers) he says will enable the diagnosis to be made during the interval. This reaction lies in the power of the blood serum of a patient who has passed through one paroxysm to agglutinate the spirochetes in the blood of a fever case, a power not possessed by normal blood serum.

Malaria is the disease for which these cases of spirochetic infection are most likely to be mistaken in this part of the world. In a patient presenting symptoms similar to malaria and who has recently arrived from foreign parts or who has been more or less intimately associated with a newly arrived person, a careful search of the blood should be made for this organism in question. It is perhaps allowable to point out that, compared with relapsing fever, malaria presents a more regular incidence of febrile periods; a shorter interval between the paroxysms, and generally a spleen more easily palpated.

The blood in malaria shows an early and a greater loss in the red cells and hemoglobin percentage. As regards the white cells, the importance of the presence of intracorpuscular pigment is to be kept in mind. The leucocytosis observed by many in relapsing fever is unusual, if not wanting, in uncomplicated cases of malaria.

On the other hand, the normal or diminished white-cell count associated with a very high relative increase in the larger lymphocytes and mononuclear cells seen in our cases, has been seen by

many in malaria (see Stephen's and Christopher's note quoted in Mannaberg's article in Nothnagel's *Encyclopædia*).

Mannaberg quotes Mamourski as having seen a case which showed both a malaria parasite and the spirochete of Obermeier in the blood at the same time.

Influenza is suggested by the sweating and the marked subnormal range of temperature characteristic of the intermission. Rabagliati says he knows of no other diseases in which this feature is so apt to be emphasized as relapsing fever and influenza. In the latter disease a relapse, if it occurs, follows the first febrile period after an apyrexial stage of much shorter duration than that that rules in relapsing fever, and the symptoms in such a case do not promptly or entirely disappear on the fall of temperature.

Considering the fact that the vomiting of blood, so common in yellow fever, may occur in spirochetic infection (Biada's Case), and that epistaxsis and jaundice occur in the latter condition, one should always look for this organism in the blood of cases showing some of the characteristics of yellow fever. A statement similar in effect may be made of cases that suggest the presence of plague, typhus fever, or sepsis.

The duration of Case I. was 30 days, of Case II., 25 days; Dutton's case lasted for 47 days, Todd's case for 42 days. Taking such an instance as Manson's Gibraltar case and adding an incubation stage of a week, we have a period of three months and in that length of time one of these cases might travel from the remotest parts. A sporadic case of spirochetic infection may, therefore, appear anywhere at any time, and undoubtedly it occasionally so happens.

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PLATE 6.

These photographs (1 500 diam.) were taken from one and the same specimen of blood from Case I.

Fig. 1.—A large spirochete.
Fig. 2.—Two spirochetes seemingly end to end.
Fig. 3.—Two small spirochetes intertwined.

FIG. 1.

FIG. 2.

FIG. 3.

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STUDY OF A SPIROCHETE OBTAINED FROM A CASE OF RELAPSING FEVER IN MAN, WITH NOTES ON MORPHOLOGY, ANIMAL REACTIONS, AND ATTEMPTS AT CULTIVATION.*

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INTRODUCTION.

The spirochetes studied were obtained from the blood of a patient in the service of Dr. Carlisle, in Bellevue Hospital.† By inoculation of monkeys and rats we have been able to keep the spirochetes alive through the continuous passage of blood from one animal to another. It is our purpose to record as briefly as possible the various observations which we have made upon the animal reactions, with a brief description of the morphology of the micro-organism, and the various attempts made to cultivate it outside the body. Our studies have continued over a period of five months.‡

Spirochetal Infection in Monkeys.

Three monkeys of the species *Macacus Rhesus,* and two of the species *Macacus Javanus,* were used for inoculation experiments. These species were selected because of their known susceptibility to infection with *Sp. Obermeieri.* The disease was produced by subcutaneous inoculation of human or animal blood containing spirochetes.

Incubation Period—Following the injection there was noted an incubation period during which no spirochetes were found in

* Received for publication, February 7, 1906.

† Dr. Carlisle's clinical report of this case is found in this number of the Journal.

‡ Condensed reports of our work were given at the October and December meetings of the New York Pathological Society, and at the Society of American Bacteriologists at its December meeting of 1905, through the courtesy of Professor Novy.

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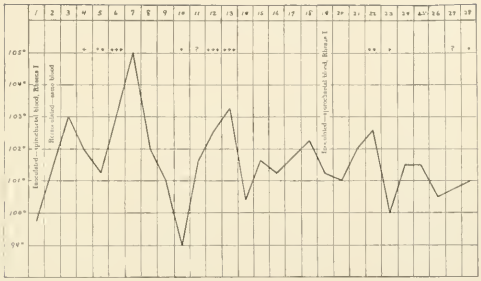
the circulating blood. The duration of this was, in one monkey (Rhesus I.), two days; in another (Rhesus III.), three days; in a third (Java II.), three days; in the remaining two, impossible to determine, because of double inoculations, but not exceeding three days in one (Java I.), or six days in the other (Rhesus II.). During this incubation period no obvious signs of illness were observed.

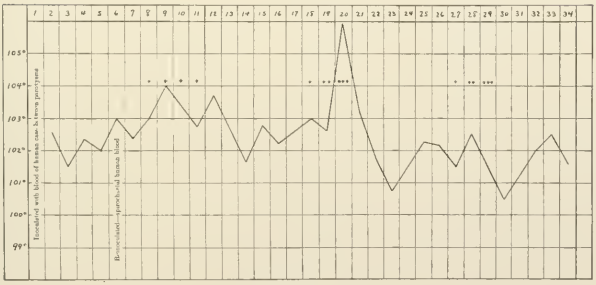
Relapses—The disease, in monkeys, like relapsing fever in man, was characterized by a succession of paroxysms, each attended by the appearance of the spirochetes in the peripheral blood, and certain general symptoms of illness. Three of the monkeys showed three paroxysms; one monkey, four. The duration of the individual paroxysms, and of the intervals, varied widely. The longest period during which spirochetes were present in the blood on successive daily examinations was four days; the shortest, one day. The average duration of the paroxysms apparently slightly decreased with the progress of the disease. The longest interval noted was eight days; the shortest, two days. The average length of interval in general appeared to increase after the first. One marked exception to this was noted, the intervals being two, eight, and three days respectively. We see, therefore, that there is little regularity or uniformity in the onset, number, or duration of the paroxysms.

General Symptoms—A rise in temperature usually accompanies the appearance of the spirochetes in the blood, and persists until their disappearance. The temperature charts, however, are far less striking than those of relapsing-fever patients. There is normally considerable variation in the body temperature of different monkeys, and even from day to day in the same monkey. Thus Rhesus I., before the onset of his first paroxysm, showed a temperature ranging from 101° to 103° F.; Rhesus II., a temperature ranging from 99.8° to 102°; Rhesus III., a temperature between 99.6° and 104.2°; and Java I., between 99.8° and 102.8°. Moreover, the febrile reaction during the paroxysm, though usually present, is poorly marked and inconstant as contrasted with the typical fever curve in relapsing fever of man. It rarely exceeds 2° F. above the normal average. The highest recorded temperature is 106°, noted in Rhesus I. toward the end of his second paroxysm.

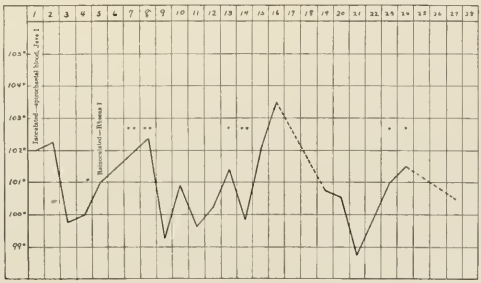
The appended temperature charts show the general course of the fever. Their most impressive feature is the marked irregularity.



JAVA I.



RHESUS I.



RHESUS II.

During the paroxysms the animals exhibit certain unmistakable signs of illness. When left alone, they sit huddled up against the wall of the cage, and from time to time cry shrilly, as if in pain, and, when disturbed, show an unwonted irritability. The appetite is poor during the attacks. In two monkeys there was a sharp diarrhea, lasting a few days. One monkey showed distinct, though transient, jaundice of the conjunctivæ. Edema of the eyelids was seen in Rhesus I. during the latter part of his illness, and Java I. developed a marked conjunctivitis of the right eye, with hemorrhage into the anterior chamber, during his second relapse.

Unfortunately, lack of time prevented us from carefully studying the blood changes during and after the paroxysms.* No differential or total leucocyte counts were made. Certain changes, however, attracted our attention in the routine examination of the blood smears for spirochetes. Very striking was the polychromatophilic degeneration of the red cells, which was constantly seen after the first appearance of the spirochetes in the blood. Granular degeneration, on the other hand, was not observed. The large mononuclears impressed us as being in excess, although without accurate differential counts, their exact numerical relation to the presence of the spirochetes in the circulating blood was not determined. Numerous very large forms, with pale, poorly staining nuclei, and ragged degenerated cytoplasm, were always present. The blood plaques appeared to be markedly increased.

The course of the disease in all monkeys was benign. The animals speedily regained their usual good health after the last paroxysm. During their illness they appeared to lose moderately in weight, especially Rhesus I. and Java I.

Spirochetal Infection in White Rats.

Up to the time of writing, about 100 rats have been inoculated. Two rats were inoculated on October 24, from Rhesus III.; since this time the spirochetes have been kept alive by constant passage from rat to rat. From a few drops to 1.5 c.c. of blood, to which sodium citrate was added, was injected beneath the skin of the back. In all cases where the inoculated blood showed spirochetes at the time of injection, a spirillosis developed

* The blood of each monkey examined before inoculation was found free from hematozoa or other blood parasites.

in the experimental animal. The only exceptions to this observation were noted in two diseased rats, which died on the second day after inoculation, and showed encapsulated abscesses of lung, chronic sepsis, and advanced secondary anemia.

The incubation period ranged from one to five days, averaging about two and one-half days. It has diminished with the frequent passage from rat to rat. Thus the average time for the development of the disease in the last 15 rats inoculated was only about day, as compared with three days in the first 15 animals used. The minimum incubation period was not determined with accuracy, because the majority of the rats were not examined until the third day. In Rat 89 numerous spirochetes were seen 19 hours after inoculation.

Owing to the fact that most of the rats were killed as soon as numerous spirochetes were found in the blood, the duration of the illness was determined in comparatively few. In those allowed to live, spirochetes were found in the peripheral circulation for a period varying from one to five days; most frequently, one to two days. It is an interesting fact that the single rat, killed after they had persisted for five days, showed multiple large chronic abscesses of the lung, from which we may infer, if we like, that his normal resistance was diminished.

No relapses were observed in two rats, whose blood was carefully studied for 12 and 18 days respectively. That relapses may not occur we cannot say definitely. The rats at no time betray any definite objective indications of illness, save the presence of the specific organisms in the blood. The same qualitative changes in the appearance of the blood cells—marked polychromatophilia, and large mononuclears, resembling bone-marrow cells, megakaryocytes—were noted as in the monkeys.

The only gross pathological change seen in animals killed during the height of infection was swelling of the spleen. No report of the histological examination of the organs will be attempted here. We must, however, state that repeated examinations of smears from the spleen, bone-marrow, and liver, stained by Wright's, Giemsa's, and other methods, failed to show spirochetes, either within or without the cells, at any stage of the disease, with the exception of a few extracellular single forms in one spleen smear. The large mononuclears, with vacuoles, we have seen in abundance. The vacuoles contained only granular detritus; nothing in the least suggestive of spirochetes. It is

possible that, with the particular organism studied, phagocytosis, if it occur at all, plays an altogether unimportant rôle. Shortly after the disappearance of the spirochetes from the circulating blood, spleen smears were likewise examined without success. This is in marked contradiction to the findings of Metchnikoff,[1] Soudakewitch,[2] Sawtchenko and Melkich,[3] with the spirochetes of Obermeier. In splenic smears from artifically infected monkeys, spirochetes were distinctly seen and depicted within the polymorphonuclears (Metchnikoff) or the large mononuclear macrophages (Soudakewitch); in the latter they often lie coiled up within a vacuole in the cell protoplasm. Sawtchenko and Melkich observed a similar phagocytosis on the part of the endothelial cells, when spirochetal blood was injected into the peritoneal cavity of guinea-pigs.

Cantacuzène[4] in the spirillosis of geese Levaditi,[5] and Marchoux and Salimbeni,[6] in the spirillosis of fowls, likewise describe the phagocytosis which occurs in the spleen toward the end of the attack. In the drawings of the spleen smears by Cantacuzène a number of clearly defined, unmistakable spirochetes are shown lying coiled within the vacuoles.

Sawtchenko, in a monkey whose spleen had previously been removed, observed that the spirochetes became very numerous during the course of infection, and persisted, a chronic spirillosis being established, the animal finally dying. The spirochetes are shown in great numbers in the blood smears taken from the superior vena cava, and within the polymorphonuclears of the peripheral blood. Such pictures were not seen during the natural course of infection in our human cases, or in the monkey or rat.* Soudakewitch[2] and Iwanoff[7] noted the occurrence of phagocytosis in the circulating blood of patients with relapsing fever, near the crises, their statements varying in this respect from those made by other observers. Excision of the spleen is not followed in all cases by a chronic spirillosis, as shown in one of our rats (90), which was splenectomized 24 hours before inoculation. In

* In the last edition of one of the noted American textbooks upon the clinical examination of the blood, we have been surprised to see the drawings and cuts of Sawtchenko reproduced, without any reference to their source, or any explanation that they were not drawn from human, but from the blood of the splenectomized monkey. In view of the fact that the spirochetes are never present in the blood of human beings or monkeys in such abundance, and that the polymorphonuclear leucocytes in the circulating blood, according to most observers and ourselves, do not contain spirochetes, a most erroneous impression of the blood picture of relapsing fever is thereby conveyed to the reader.

this case the infection ran the usual course, the spirochetes appearing and disappearing one day later than in the control rat.

SPIROCHETAL INFECTION IN RABBITS.

Animal Inoculated.	Source of Infectious Material; Dose; How Inoculated.	Remarks.
Rabbit III., Sept. 19	1 c. c. of citrate blood from Java I., intravenously. (Smears positive)	Few spirochetes on following day; numerous spirochetes on second day; negative on succeeding days. No spirochetes in smears from liver, spleen, or bone marrow.
Rabbit IV., Sept. 19	0.5 c.c. of citrate blood from Java I., intravenously.	Few spirochetes on following day; negative on succeeding days.
Rabbit V., Sept. 21	1 c.c. of citrate blood from Rabbit III., intravenously. (Smears sp. ++)	Negative.
Rabbit VI., Oct. 2	0.5 c.c. of citrate blood from Rhesus I., intravenously. (Smears sp. ++)	Negative.

As seen from the above table, two of the rabbits showed undoubted susceptibility to spirochetal infection, if we are to judge by the multiplication of the spirochetes in their blood. The animals did not appear ill, and the organisms persisted for only one or two days. There were no relapses. The one attempt to inoculate a second rabbit with the spirochetal blood of Rabbit III. was unsuccessful. It may be remarked here that many articles on relapsing fever state specifically that the rabbit is not susceptible to infection with the *Sp. Obermeieri*, and we have met with no previous record of successful inoculations in rabbits, except that of Dutton and Todd.[8] This discrepancy may possibly be explained by the fact that young rabbits were used, and the material was injected intravenously. It is obvious, however, that rabbits are not well adapted for experimentation with the spirochetes, and in our further work white rats were used exclusively, because of their constant susceptibility and lesser cost.

SPIROCHETAL INFECTION IN GUINEA-PIGS.

Several guinea-pigs inoculated intraperitoneally failed to reveal spirochetes in the circulating blood.

SPIROCHETAL INFECTION IN WHITE MICE.

Two small white mice were inoculated with spirochetal blood from Rat 80. On the second day numerous spirochetes were found in the blood. No further observations on mice have been made.

Immunity.

A. *Active Immunity*—Both in rats and monkeys previous infection confers immunity. We are unable to say how long this immunity persists, since our observations cover a period of only three or four months. We do know, however, that it has lasted for 94 days in the case of Rhesus I., 86 days in Rhesus II., and 64 days in Rhesus III. Inoculations with spirochetal blood (Rat 78) after these intervals were negative. The control monkey (Java II.), inoculated with the same blood from Rat 78, showed spirochetes in the blood on the third day. Only four observations have been made on rats, but those animals likewise could not be reinfected. Rats 4, 6, 41, and 46 were reinoculated after periods of 13, 10, 41 and 33 days respectively, following the last appearance of the spirochetes in their blood. Daily examinations for seven to eight days failed to show spirochetes.

B. *Passive Immunity*—In contrast to the perfect protection acquired through previous infection is the uncertainty of the protection furnished by the inoculation of serum from man and animals that have passed through a previous infection. The poor results obtained in our experiments upon the production of passive immunity are doubtless due to the fact that the serum used was not obtained from actively immunized animals—a statement in accord with the observations of Professor Novy,[9] who found that the injection of the blood from actively immunized rats not only prevented the infection in white rats, but caused the rapid disappearance of the spirochetes from the circulating blood.

We submit a synopsis of our experiments along this line, and the inferences which in our opinion may reasonably be drawn from them.

a) In these experiments serum and spirochetal blood were mixed before inoculation.

Experiment I.—Rat 22: Inoculated with 1 c.c. of blood from Rat 16, and 1 c.c. of normal human serum (kept five days at room temperature); in contact for five minutes before inoculation. Spirochetes appeared on the third day in considerable numbers.

Rat 24 (control): Inoculated with 1 c.c. of blood from Rat 16. Spirochetes appeared on the third day.

Rat 21 (control): Inoculated with 1 c.c. of blood from Rat 16. Spirochetes appeared on the third day.

Normal human serum exerted no inhibitory power upon the development of the infection.

Experiment II.—Rat 23: Inoculation with 1 c.c. of spirochetal blood from Rat 16+1 c.c. of human serum (A. M. P.) obtained 12 days after second and last relapse. (Serum kept for five days at room temperature.) Spirochetes appeared on the fifth day.

Rats 21, 24 (controls): See Experiment I.

The "immune" human serum apparently caused a retardation of the infection for two days.

Experiment III.—*Rat 25: Inoculated with 1 c.c. of blood from Rat 16 + 1 c.c. of serum from Rhesus I., obtained 43 days after the third relapse, and 15 days after inoculation with spirochetal blood from Rat 1. (Serum kept 24 hours at room temperature.) Blood negative for nine days; not further examined.

Rats 21, 24 (controls): See Experiment I.

Complete protection was afforded in this case by the serum of a monkey after natural infection, and after a single subsequent inoculation with spirochetal blood.

b) In the following experiment the immune serum was injected previous to the inoculation with infective blood.

Experiment IV.—Rat 26: Inoculated subcutaneously with 20 gtt. of serum from Rhesus I., drawn 43 days after the last relapse, two days old. Three days later inoculated with 15 gtt. of spirochetal blood from Rat 21. Spirochetes appeared on the third day.

Rat 30 (control): Inoculated with spirochetal blood from Rat 21. Spirochetes appeared on second day.

c) In the following experiment an unsuccessful attempt was made to cause the disappearance of the spirochetes from the blood during a paroxysm, by the injection of the citrate blood of a rat which had passed through its infection. We were led to perform this experiment by the very striking and favorable results recorded by Professor Novy[8] in the use of blood from *actively* immunized rats.

Experiment V.—Java II.: Inoculated with spirochetal blood from Rat 78. A few spirochetes appeared on the third day, and were very numerous and extremely motile on the fourth day. On the fourth day the monkey was inoculated subcutaneously with 5 c. c. of blood of Rat 75, withdrawn five days after last appearance of the spirochetes. Blood was drawn from the ear and examined every 15 minutes for one hour, and again after two, three, and five hours.

No effect was produced, the spirochetes becoming more numerous and retaining their motility after five hours. On the following day, 24 hours later, no spirochetes were found. The first relapse occurred five days later.

* A similar set of experiments was performed five days previously. The results are not given in detail, because two of the rats escaped from their cages, and could not be positively identified. In this case the immune human and monkey serum afforded protection, whereas control rats and rat inoculated with normal human serum developed spirochetes on the third day.

It is evident from this experiment that the anti-bodies in the blood of a rat which has recovered from the spirochetal infection are not present in quantities sufficient to abort the infection in other animals. Furthermore, the inoculation of this serum did not inhibit or retard the onset of the first relapse, or shorten its duration.

It may be of interest to note an additional observation made at this time. Immune blood of the above rat (No. 78), as well as the immune blood of Rhesus III., when mixed with the spirochetal blood of Java II., and examined in hanging-drop, showed no agglutinating properties, nor was any decrease in the motility of the organisms observed, after several hours. (Apparent lack of "immobilisines" of Levaditi.) The only phenomenon noted was a marked tendency of the spirochetes to adhere to the red blood corpuscles. In the hanging-drop preparation, after 24 hours at room temperature, no spirochetes were found, however, whereas in the control hanging-drop, and with mixtures of Java II. and Rat 75, numerous clumps of spirochetes were seen, only a few of the organisms being still motile. The dissolution or cytolysis of the spirochetes evidently took place slowly in serum.

Morphology.

Because of the emphasis that has been laid upon the morphology of the spirochetes as a basis for their classification, by Schaudinn[10] and other writers, it seems worth while to enter into a somewhat minute description of the forms which the organisms studied by us may assume.

Under conditions of which we know practically nothing, these spirochetes may show the most astonishing variations—variations in length, in thickness, in the number and closeness of the spirals, in motility, and in arrangement.

The spirochete, as seen in smears, is a tapering, spiral filament, whose length varies roughly from one and one-half to 10 times the diameter of a red cell; exceptionally, even longer forms are seen. The breadth or thickness is more constant; but still one meets with very slender (attenuated) forms, on the one hand, and thick, almost ribbon-like or fusiform types, on the other. In some rats we have observed that the breadth varies markedly in different portions of one and the same spiral. The number and conformation of the spirals are subject to very great variations. The conditions which determine these variations in morph-

ology we have not been able to ascertain positively. These marked variations are observed only when the spirals are most numerous; that is, within 24 hours previous to their disappearance from the circulating blood. The extremes are typified by a short organism, showing but a single sinuous curve; and a long one with 15 or more closely approximated spirals. Between these two there may be found every transitional form.

The tapering ends of the spirochetes in successful smears are usually drawn out to a considerable length, so that they may readily be interpreted as terminal whips or cilia. In special stains for cilia we have never seen more than one terminal filament.

In every stained preparation examined the majority of the spirals show in their central portion an unstained area, where the substance of the spirochete is of smaller caliber, as if it were thinned out. In long forms, two, occasionally three, attenuated portions may be seen. This attenuation may be so marked at one point that it is difficult to say whether the continuity between the two portions is preserved or broken. Numerous spirochetes may also be found, lying end to end, with their tapering extremities in close approximation, strongly suggesting a recent separation.

These appearances, to our mind, force the conviction that reproduction in these spirochetes normally occurs by transverse fission, or possibly fragmentation. In spite of painstaking study, we have not succeeded in actually seeing this take place in living preparations. Because of their active motility, their low refractive index, and the difficulty in keeping the entire spiral in one optical plane, it is practically impossible to watch the same organism continuously for any length of time.*

In stating that the spirochetes multiply by transverse division, we do not exclude the possibility that other modes of reproduction may take place. Of longitudinal division, or of sporulation, we have seen no evidence.

When the spirochetes are abundant, one frequently finds two or more organisms entwined, or, when very numerous, in tangled masses. They are, however, so often seen in certain definite relations to each other that one might be inclined to attribute to this a more than casual significance. We have been impressed with the frequency with which two spirochetes are seen entwined

* On one occasion transverse division was observed, but, owing to the difficulty of being positive in the interpretation of such phenomena, the above conservative statement is advisable.

throughout their entire length, or lying side by side, accurately apposed, or even apparently fused through a portion of their length. The fused portions may terminate as a broad mass, ending in several short terminal filaments. We do not think that this appearance signifies a longitudinal division. These appearances remind one of the fusion which has been observed in the bacteria; or, again, it may be simply an expression of their tendency to adhere to each other under certain conditions. The spirochetes tend to adhere to the red blood cells, to the hemokonia or blood dust of Müller, to the blood plaques, and to various particles present in the blood. Finally, as a theoretical possibility, one may think of a conjugation analogous to that seen in the protozoa.

Extraordinary forms have been noted in the blood of a few rats, which require notice. In Rat 87 the spirochetes were found in great numbers. Many of the tangled masses were composed of individuals having the usual spiral appearance. Other masses were composed of more or less flattened or ribbon-like, and partly sinuous or even straight, individuals, these varied forms being more or less intimately united by faintly staining intercellular substance. Ribbon-like forms were quite common. In some of these thick forms an intervening clear space was made out, with the aid of compensation ocular No. 8. (See Plate 7, Fig. 4.) The ribbon-like forms may stain quite intensely, although usually they show a pale-blue color with the ordinary blood stains. Forms are not infrequently seen in which most of the protoplasm stains faintly, with several more deeply stained areas. The appearance occasionally suggests the presence of a macro- and micro-nucleus, but, on the whole, the more acceptable, and less strained, explanation is that, as in bacteria, irregularity in staining occurs. To us the ribbon-like forms are analogous to the so-called involution or degeneration forms of bacteria. The peculiar forms above described we have seen in rat blood, when the spirals are numerous, just before their final disappearance from the circulating blood. Under such conditions, it seems more reasonable to infer that these peculiar forms are the expression of degenerative or necrobiotic changes rather than developmental changes or evidences of longitudinal division.

Staining Reactions.

Films have been stained with Wright's, Jenner's, Giemsa's, and Goldhorn's blood stains; with the ordinary aniline dyes, and with

Gram's stain, the spirochetes decolorizing by the latter method; also with Loeffler's and with Proca's[11] flagella stain.

Wright's stain has been used for routine examination. With this stain the spirals are colored a blue of moderate intensity, or, if the differentiation is carried farther, a faint or even a pronounced red. The other polychrome stains have yielded, in our hands, no essentially different results. Hematoxylin stains them faintly, but uniformly.

With methylene blue, followed by Gram's iodine, the spiral is stained grayish black throughout its entire length, this being the color which the metachromatic granules of bacteria assume when stained by this method. The results obtained by the use of these stains—that is, a uniform, diffuse stain—speaks strongly against the assumption of the presence of a macro- and micro-nucleus. By means of aqueous methylene blue an intra-vital staining may be brought about in the hanging-drop, the spirals retaining their motility for 24 hours or longer.

Motility.

In fresh hanging-drop preparations the most characteristic movement is a very active wavy or corkscrew one, broken by an asystolic interval of variable length.* This movement may apparently start from either end, and an alternation in the wave of contraction is always noted, except when a spiral becomes attached by one end to some object. A very definite lateral oscillation also takes place. This ranges from a slight trembling to a violent to-and-fro agitation of the entire spiral. At times a distinct and rapid forward-and-backward progression is seen, the total excursion sometimes covering half the microscopic field of an immersion lens.

In the hanging-drop kept for two days, either at room temperature or in the incubator, the spirochetes retain their various forms of movement. As their vitality becomes impaired, however, their movements grow less rapid, and often assume a tetanic character. When the corkscrew movements become slow, and occur at longer intervals, it can readily be made out that the motion in one direction originates at one extremity, the other extremity determining motion in the reverse direction.

* For very simple optical reasons, it is impossible to decide whether this is an actual revolution about the long axis, or a rhythmic waving in one plane, comparable to the waving of a pennant in the wind.

Degenerative and Necrobiotic Changes.

With this gradual loss of motility the spirochetes undergo a marked change in appearance. The threads become swollen and unevenly refractive, resembling the thick, ribbon-like forms found in stained blood smears. Later there are constantly found tenuous, beaded forms, which may be compared to some of the irregularly staining or moniliform individuals seen in the blood films. The presence of such forms only shortly precedes the final disappearance of the spirals from the citrated blood.

It is our impression, therefore, that these forms represent a stage in the dissolution of the spirochetes.

Attempts at Cultivation.

Many attempts have been made to obtain cultures of the spirochetes, all of which have been unsuccessful. Basing our choice of media upon a possible relationship between these organisms and the trypanosomata, the 2 to 1 rabbit blood agar as recommended by Novy and McNeal[12] was tried. This medium, though tested a number of times, gave no results.

Various attempts were made to obtain cultures in fluid media. These media may be briefly mentioned: human blood with meat extract broth; human blood with human muscle broth; human blood with rabbit muscle broth; ascitic broth with and without blood. Inoculations on these media were negative. In some cases the blood, before being added to the broth, was mixed with sodium citrate solution, and in others the medium containing blood was heated for one-half hour at 55° C., to destroy the complement.

In some cases the blood was laked by citric acid, and subsequently neutralized by the addition of sodium carbonate.

Cultures were kept both at room temperature and in the incubator. In none of these fluid media was any evidence of multiplication observed, the spirals never being recovered.

Evidences of multiplication, however, were obtained in the condensation water of human and rat blood agar, the agar being made up with 2 per cent. peptone and 1 per cent. dextrose, and with human muscle infusion instead of the usual Liebig's meat extract. The condensation water, after 24 hours at room temperature, showed actively motile spirochetes in the hanging-drop, in greater numbers than in the original blood, as determined by the number seen in smears of the blood made at the time of inoculation. Transplants of a few loops from the condensation

water to similar blood-agar tubes yielded practically negative results, although in many cases a few spirals were found on examination. The tubes kept at 37° C. uniformly gave negative results.

Evidences of Multiplication of the Spirochetes in Human and Rat Citrate Blood at Room Temperature.

Positive evidences of the multiplication of the spirochetes in fluid media were observed in a number of experiments. In human and rat blood, to which has been added sodium citrate to prevent coagulation, there can be seen, within 24 hours after inoculation with a few drops of rat blood containing spirochetes, a very evident increase in the number of these organisms. By this we mean that, notwithstanding the dilution of several drops of infected blood with 60 to 150 times its volume, the spirochetes are more numerous in the smears from the culture fluid than in control smears taken at the time of inoculation.

Furthermore, after inoculating several drops of this first generation into a second blood tube, we have found the organism in approximately the same numbers in the second transplant.

The third generations, however, have failed to grow, although in the hanging-drop, non-motile, and in stained specimens, granular or moniliform organisms were seen. Although multiplication of these organisms in citrate blood was the rule, in a few cases increase was not observed. For instance, citrate blood, obtained from some patients, seemed to exert a deleterious influence upon the spirochetes, none being found in the blood after 24 hours. These variations in human blood may account for some of the negative observations. Multiplication likewise occurs in the undiluted citrate blood from infected rats, kept over night at room temperature, and in many cases tangled clumps develop.

The following brief description must suffice to justify the above statements upon the occurrence of multiplication:

Rat 51: December 4, inoculated with citrated blood of Rat 49, showing spirochetes. December 7, a few spirochetes found in stained smears. Rat was then etherized, and blood aspirated from heart. A few drops of blood introduced into a test tube containing 5 c.c. of human citrated (circa 3 per cent. sodium citrate) blood. Tube kept at room temperature. In hanging-drop preparations, 19 hours after inoculation and in smears stained by Wright's stain, numerous single as well as tangled masses of spirochetes were seen. The drops for examination were taken from the surface of the somewhat turbid supernatant fluid. Transplants of a few

drops from this tube into human citrate blood, on examination after 24 hours at room temperature showed actively motile spirals, not as numerous as in the first generation. Third generations were practically negative.

An agar flash containing 20 c.c. of Medium H* and 2 c.c. human citrate blood inoculated with a drop of infected rat blood, and kept for 19 hours at room temperature, showed numerous spirochetes in hanging-drop.

Rat 54: December 6, inoculated with blood of Rat 50, latter showing a few spirochetes. December 9, a few spirochetes found in smears made from blood of rat's tail; rat etherized, heart's blood aspirated, and mixed with citrate solution, and a tube containing 3 to 5 c.c. citrate human blood was inoculated with two drops of blood. After 24 hours at room temperature, the rat blood diluted with citrate solution showed numerous single and tangled masses of spirals in hanging-drop and in smears from fluid taken from surface of the settled blood tube. Transplants from this tube in sterile citrate rat blood were unsuccessful. In the citrate human blood tube no spirals were found. Plain human meat broth, and two tubes of human meat broth 5 c.c.+1 c.c. human citrate blood, inoculated with several drops of blood of Rat 54, show no spirochetes on examination.

In this experiment there was an evident multiplication of the spirals in the original rat blood, no multiplication being observed in the dilutions.

Rat 55: As in Rat 54, there was an evident multiplication of the spirals in the original citrate rat blood after 24 hours at room temperature. No evidence of growth occurred in the citrate human blood tubes inoculated with a few drops of rat blood.

Rat 56: December 9, inoculated with blood of Rat 54, latter showing numerous spirals. December 11, extremely numerous spirals were found. Citrate human blood (3-5 c.c.) tubes, and normal rat citrated blood (3-5 c.c.), were inoculated with several drops of the heart's blood of Rat 56, and the rest of the blood was citrated and kept at room temperature. After 18 hours the spirochetes had multiplied greatly in the original citrated blood (Rat 56). Many large clumps and numerous single spirals were seen in the hanging-drop and in stained smears made from the fluid. The normal rat and the normal human blood likewise showed extremely numerous and actively motile spirochetes. The spirals were found not only on the surface, but also in equal numbers at different levels of the fluid, proved by carefully drawing off the fluid in fine capillary pipettes. Transplants to normal rat blood showed in hanging-drop a few slightly motile, but beaded spirals. Transplants from this second generation remained negative.

This experiment indicates that in the first generation in normal rat blood rapid multiplication takes place, but that further growth is for some

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* Medium H:

Peptone	2 per cent.
Sodium chloride	5 " "
Dextrose	2 " "
Agar	2 " "

Slightly alkaline to phenolphthalein.

reason inhibited. It has been constantly observed that where laking of the citrated blood occurs, as in the second-generation tubes of this experiment, no multiplication takes place.

Rat 57: December 9, inoculated with blood of Rat 54. December 12, a few spirals found in one large smear. Rat etherized, blood citrated, and a few drops inoculated into human citrated blood. After 18 hours the spirochetes were found in considerably greater numbers in the culture tubes than in tubes containing the original citrated blood.

Transplants of a few drops to tubes of the same medium showed, after 24 hours, a few motile and non-motile spirals in hanging-drops and in smears. Transplants from these tubes were unsuccessful.

Rat 58: December 11, inoculated with blood of Rat 57. December 14, spirochetes found. Smears of the blood at time of etherization of rat showed two spirochetes after five minutes' search. Rat etherized, and blood inoculated into tubes, which, 24 hours later, gave the following results:

Tube 1,	normal	citr.	rat	blood,	inoc.	with	2 gtt.	Sp. not found.
" 2,	"	"	"	"	"	"	2 "	Sp. moderate number.
" 3,	"	"	"	"	"	"	2 "	Sp. numerous.
" 4,	"	"	"	"	"	"	2 "	Sp. few.
" 5,	"	"	"	"	"	"	4 "	Sp. not found.

(heated 30′ at 56° C.)

Tube 6, normal citr. rabbit blood, inoc. with 4 gtt. Sp. not found.

Transplants were made as follows:

From tube 3: a few drops into { citrated rabbit blood—2 tubes. " rat " 1 tube.

From tube 2: a few drops into { citrated rabbit blood—2 tubes. " rat " "

After 24 hours at room temperature, transplants from Tube 2 showed a few straight, typical, slightly motile and non-motile forms; while the original citrated blood (Rat 58), examined on the same day (48 hours), showed numerous active spirals. Transplants from Tube 2 negative.

The extreme scarcity of the spirochetes in the blood at time of killing of rat, when compared with numerous spirals found in the above blood tube at 48 hours, as well as in transplants, indicates active multiplication. In this experiment the rapid multiplication observed is confined to the tubes of the first generation. Something inimical to the growth and even preservation of the spirochetes makes its appearance.

Rat 59: December 11, inoculated with blood of Rat 57. December 14, spirochetes numerous. December 15, spirochetes numerous, rat etherized, several drops of blood inoculated into:

3 tubes of normal citrated rabbit blood.
3 " " " " rat "

Cultures on the three rabbit blood tubes were negative, when examined 24 hours later. A moderate number of spirals were found in hanging-drops from the rat blood, and in one of these tubes (Tube 6; see Plate 7, Fig. 6), clumps of actively motile spirochetes were found after 24 hours.

Several loops of this tube transplanted into citrated rat blood, revealed six actively motile spirals in one hanging-drop, which, when the dilution is taken into account, may indicate only the number actually carried over.

Rat 64: December 17, inoculated with blood of Rat 62, latter showing numerous spirochetes. December 20, very numerous single spirochetes, and also small clumps found in stained smears.

Cultures made on three tubes of citrated normal rat blood, each tube being inoculated with one drop of spirochetal blood. Tubes examined after 24 hours at room temperature.

Tube 1, a few motile spirochetes found.
" 2, no spirochetes found (slight laking).
" 3, not examined " " .

Tubes after 48 hours at same temperature:

Tube 1, spirochetes in moderate numbers.
" 2, only one spirochete found in hanging-drop.
" 3, spirochetes in moderate numbers.

This experiment has been introduced for the purpose of demonstrating that the best cultural results (indicated by multiplication) are obtained with blood of rats which show few spirochetes. (See above experiment, Rat 58.)

Considering the large number of spirochetes in the blood of Rat 64, no multiplication can be claimed in this experiment, even when the dilution of the blood is taken into account (1 drop in 3-5 c.c. of normal citrate rat blood).

Rat 66: December 20, inoculated with blood of Rat 64, showing spirochetes very numerous. December 22, spirochetes in moderate numbers. Cultures in normal citrate rat blood were negative, while spirochetal rat blood, after standing 24 and 48 hours, showed numerous spirochetes, most of them motionless and in clumps.

Rat 83: January 8, inoculated with blood of Rat 81. January 10, a few spirochetes found in blood after search through many fields. Rat etherized, and two drops of blood inoculated into 5 to 6 c.c. of citrated normal rat's blood. Blood of Rat 83 citrated and distributed in two tubes. After 24 hours at room temperature, the three tubes showed extremely numerous and actively motile spirochetes, a dozen or more to the field of the hanging-drop, and after 48 hours showed clumps and spirochetes which had lost their motility. Transplants (two loops), made after 24 hours upon the same medium, show next day only a few spirochetes.

This experiment indicates not only that a multiplication of the spirals must have occurred in the original citrate rat blood, but that a much greater multiplication took place in the normal citrated rat blood inoculated with only two drops.

As an illustration that this multiplication by no means always occurs in the normal rat blood, the following experiment is given:

Rat 85: January 10, inoculated with blood of Rat 83. January 12, a few spirochetes found. Rat etherized. The citrated blood, and the normal citrated rat blood inoculated with a few drops, were examined

24 hours later. Only an occasional spiral was found in the citrated blood of Rat 85, the dilution being negative at this time and also later.

We believe that from these experiments we are justified in concluding that under favorable conditions a multiplication of the spirochetes occurs outside of the body. This conclusion is based upon the experiments detailed above, the results of which may be summarized as follows:

If a few drops of spirochetal rat blood be inoculated into 3 to 5 c.c. of citrated blood (human or rat blood), the spirochetes are frequently found in greater numbers than in the original infected rat blood kept, under similar conditions, at room temperature; and in much greater numbers than in the circulating blood when the tubes are inoculated, since at this time their discovery often requires a prolonged and careful search.

That the spirochetes found in the fluid medium represent the individuals which have been transferred with the few drops of rat blood is opposed by the fact of their being more numerous than in the original citrated rat blood, and also by the fact that they are present in approximately equal numbers at different levels of the fluid.

Only three previous observers have noted an increase in the numbers of spirochetes under artificial conditions.

Multiplication in defibrinated blood was noted by Lachmann[13] in 1880. Three experiments illustrating this are cited by him. Whereas examination of the blood when withdrawn (at the onset of the symptoms) showed spirochetes to be either absent or present only in small numbers, they appeared to increase rapidly in the blood removed, so that on the following day 3 to 20 were found in each field.

Albrecht[14] also states that he has frequently observed multiplication, but gives no detailed observations. His account of the development of the spirochetes *in vitro*, from refractile particles in the blood, has not been substantiated and is doubtless incorrect.

Gerhardt[15] has also noted an increase in numbers in blood withdrawn during a paroxysm, as well as their appearance after 24 hours in blood taken just before the onset of the attack, at a time when no spirochetes were found.

All other observers, on the other hand, have denied the occurrence of this multiplication. (See Kolle u. Wasserman.[16])

General Considerations Upon the Identity and the Biology of the Spirochetes Above Described.

A discussion and review of the literature of this class of micro-organisms, the spirochetes, would extend the limits of the article unnecessarily. Moreover, this topic has been excellently and fully presented by Wladimiroff,[17] and quite recently by Waldemar Löwenthal,[18] while references to the recent articles are found in the publication edited by Schaudinn.[19]

However, a few words may not be amiss in calling attention to the recognition of a certain number of new diseases in man and animals as being caused by species of this interesting class of micro-organisms. Twenty-three years after the demonstration of the spirochete of relapsing fever by Obermeier[20] (1868; observation published 1873), Sakharoff[21] (1891) demonstrated the etiological connection of *Spirocheta anserina* with septicemia of geese. Marchoux and Salimbeni,[6] in 1903, published their observations upon the Brazilian disease of fowls, and have likewise established another spirochete, the *Sp. gallinarum*, as the pathogenic agent of this disease. Laveran,[22] in 1902, announced for A. Theiler, the discovery of a spirochetal infection of cattle in South Africa, due to the *Sp. Theileri*. Martoglio and Carpano[23] have recorded a case of spirochetal infection in a sheep, the organism being essentially similar in morphology to the spirochete of Obermeier. By the observations of Ross and Milne,[24] Dutton and Todd,[8] of R. Koch,[25] and of other observers, the tick fever of man in Africa has been definitely decided to be due to a spirochete which greatly resembles in its morphology the *Sp. Obermeieri*.

C. Nicolle and C. Comte[26] describe briefly a spirochetal infection of bats in Tunis, which is of especial interest on account of the prevalence of relapsing fever in the Arabs of Tunis and the surrounding country. Monkeys and white mice are immune. In morphology it resembles, according to these observers, the spirals of relapsing fever. It has finely tapering extremities, stains easily, and divides by transverse division. All these spirochetal blood infections have one characteristic in common, namely, the constant presence in the circulating blood of the spirochetes during the course of the disease.

The morphology of these various spirochetes, according to different observers, is again strikingly similar. Thus, the description of *Sp. Theileri*, of *Sp. gallinarum* by Levaditi, of the spirochetes

found in bats by Nicolle and Comte, of those found in sheep by Martoglio and Carpano, and the spirocheta of tick fever described by R. Koch, are in striking accord, and are not at variance with the morphological appearances presented by our spirochete. At the present time it is only by means of the animal reactions produced by this class of micro-organisms that those producing the infections in man can be separated from those causing infections in animals. With these points in view, the *Sp. anserina* may be considered to be a species distinct from *Sp. gallinarum*.

While Dutton and Todd believe the relapsing fever of Europe and the tick fever of Africa to be identical, Koch has observed certain differences, such as the mildness of the clinical symptoms, and scarcity of the spirals in the circulating blood. As a result of his researches with the spirochete isolated by us, Professor Novy[9] believes that it is identical with the *Sp. Obermeieri*. Furthermore, he is inclined to believe that the relapsing fever of Europe and the tick fever of Africa are due to different spirochetes, on account of differences in the course of the spirochetal infection in white rats, as observed by Dutton and Todd, and by himself.

In contrast to the benign disease brought about in monkeys by the inoculation of our spirochete, the observations recorded by Dutton and Todd, and by R. Koch, show that the infections in their monkeys ran a more severe, or even fatal, course.

Infection in our monkeys has been constantly attended by the appearance of several paroxysms, while the disease caused in the same animals by the *Sp. Obermeieri* is characterized by the rarity with which relapses occur. Since inoculations in white rats with the latter organism have not been recorded, the infections of these animals described by Dutton and Todd, and by ourselves, cannot be claimed to be differential animal reactions.*

The question of the hour concerns itself with the classification of the spirochetes. Schaudinn believes that *Sp. pallida* and all other spirochetes are protozoa, and not bacteria. Vuillemin[27] has renamed the *Sp. pallida*, designating it *Spironema pallidum* on account of certain morphological appearances. This nomenclature was at first accepted by Schaudinn, but later, owing to the fact that the name *Spironema* is used in zoölogy for another form of life, he proposed the generic name *Treponema*.

* Several observers (Carter,[28] Karlinski[26]) state that mice are insusceptible; whereas Mamurowski[26] states that rats are naturally immune.

According to Schaudinn, the coils of the *Sp. pallida* are permanent—a characteristic which serves, with the demonstration of a terminal cilium, to separate it from the true spirochetes, which have more or less blunted ends. The *Sp. pallida* is differentiated by Schaudinn from the spirilla by the presence of only one cilium instead of a terminal bush of cilia, by the flexibility of the spirals, and by the occurrence of longitudinal division. Although he considers this last point not positively established, nevertheless he believes the occasional demonstration of two terminal cilia to be an indication of approaching longitudinal division. He has been able to demonstrate in the living and in stained specimens a membrane or a periblast in all species of spirochetes, except the pallida. Lately, he is under the impression that he has detected a membrane around *Sp. pallida* in the fresh condition.

Following Schaudinn's argument, our spirochetes must be classed with *Sp. pallida*. The ends are invariably drawn out, and terminate in delicate filaments, quite as long and as delicate, if not more so, than the cilia he has described and depicted for the *Sp. pallida*. These thinned-out ends he terms cilia. But, as we have stated above, these terminal attenuations differ materially from the cilia of bacteria, since, unlike the latter, they appear to be direct prolongations of the body protoplasm, and, in accordance with this, have the same qualitative staining reactions. For these reasons, it seems to us more appropriate to call the tapering extremities of our spirochetes, as well as those of *Sp. pallida*, terminal filaments.*

Furthermore, our spirochetes in rest and in motion retain their spiral coils.

We have already referred to our photographs which show the closest approximation to an indication of longitudinal division which we have observed in many hundreds of stained smears of rat, monkey, and human blood. It is not necessary to enter again into a discussion of our explanation of such appearances.

On account of the unsatisfactory evidence which the study of the morphology of this class of microorganisms has furnished.

* Since writing the above section, there has appeared in the Berl. klin. Wchnschr., 1906, No. 7, February 12, a further communication from R. Koch, relating to the African recurrent fever. In this article Koch states that he has been unable to confirm the statements of Schaudinn upon the Sp. Obermeieri. He, like ourselves, has found no evidence of a blepharoplast, nucleus, or undulating membrane (Flimmersaum). Professor Zettnow, to whom Koch has referred for support concerning the morphological evidences on these points, has made the interesting observation that the terminal filament of the spirochetes unlike the cilia of bacteria, stain with methylene blue—an observation which confirms our own.

many investigators have made attempts to determine the nature of the spirochete by a study of its biological characteristics in those insects which are known to be the active agents in the transmission of the various spirochetal infections. The first observations seem to have been made by Tictin[31] upon the transference of relapsing fever to monkeys, by the injection of the crushed bodies of bed-bugs which had fed upon the blood of fever patients. These observations prove simply that the spirochetes remain alive a few days in the bed-bug.

Marchoux and Salimbeni have infected fowls by the bites of certain insects, the *Argas miniatus*, which have fed on spirochetal blood. Theiler believes that the African cattle fever is transferred by ticks under natural conditions. Dutton and Todd have lately shown that the tick is responsible for the transmission of the human tick fever. Ticks developed from eggs dropped by infected insects transmit the spirocheta infection to monkeys. These observations, and those made by Schaudinn upon the developmental stages of *Sp. Ziemanni*, coupled with the weight of his authority, have induced many to regard the spirochetes as protozoa. A careful examination of Dutton and Todd's paper shows that they have observed no developmental stages in the tick, which would justify them in their conclusion that the spirochetes undergo a cycle. They found the spirochetes unchanged in the stomach and in the malpighian tubes. On the other hand, Koch, it seems, has definitely shown that they undergo no developmental stages in the tick, but pass out of the stomach in a few days, and reach the ovary, where they multiply rapidly, and surround the eggs in tangled masses.

Borrel and Marchoux[32] have shown that the *Sp. gallinarum* remains alive and infective for a long time in the bodies of the *Argas miniatus*, when the insect is kept at a temperature of 35° C. The spirochete is found at first only in the stomach; later it pierces the stomach wall, and may be found in the general body cavity and in the ducts of the salivary glands. No evidence was obtained of a developmental cycle or transformation of the spirochetes within the bodies of this insect. These observations are in accord with the well-known viability of the *Sp. Obermeieri*, as shown by its remaining alive in capillary pipettes and in blood kept at room temperature for several days. Pasternazki,[33] fifteen years ago, showed that the spirochetes remain alive in the body of the leech for a considerable period of time, and, more recently,

Karlinski[34] has found them alive in bed-bugs for thirty days after feeding on spirochetal blood.

The foregoing considerations as to the biological status of the spirochete, based upon our work and that of other observers appear to warrant the following statement:

The absence of any definite indication of longitudinal division; the absence of such chromatic particles as justify belief in the existence of macro- and micro-nucleus; the unquestionable occurrence of transverse fission; the positive evidence of the production of active immunity, and, as shown by Professor Novy, the formation of antibodies in sufficient amount to lend passive immunity: these facts, the authors believe, indicate that the spirochetes must not be considered as protozoa.

On the contrary it would seem more rational, at the present time, to consider them to be either bacteria or in a class by themselves.

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EXPLANATION OF PLATE 7.

Fig. 1.—Usual type of spirochete. Human blood, Case I. (Wright's stain, ×1,500.)

Fig. 2.—Rat 73. Two short forms, each with three spirals. The lower is greatly thickened. (Wright's stain; ×1,500.)

Fig. 3. Rat 87. Numerous long forms, joined end to end, or partially approximated. Beginning agglutination (?). In the right upper quadrant two spirochetes are seen, closely opposed throughout the greater portion of their length. (Goldhorn's stain;* ×2,000.)

Fig. 4.—Rat 87. Very long form, consisting of three portions, separated by attenuated intervals. The upper and middle portions are considerably thickened, and ribbon-like. There appears to be a small, unstained cleft in the upper portion. The middle segment shows two deeply staining points, where the flattened spirochete is turned upon itself. (Goldhorn's stain; ×2,000.)

Fig. 5.—Rat 87. Two long spirochetes, end to end, apparently having recently divided. Each spirochete has a thinned-out central portion. Here is a deeply staining granule on one of the organisms. (Goldhorn's stain; ×2,000.)

Fig. 6.—Rat 59. Tube 6. (Citrated normal rat blood; first generation, 48 hours.) Smeared with addition of ascitic fluid. Tangled clump showing tendency to radial arrangement. Moniliform degeneration of individual spirochetes. (Wright's stain; ×1,500.)

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* Goldhorn's recent modification of his polychrome methylene blue stain, described at the January meeting of the New York Pathological Society, 1906.

FIG. 1.

FIG. 2.

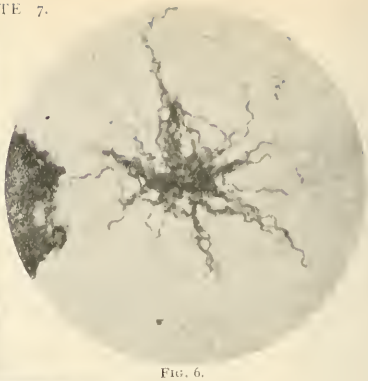

FIG. 6.

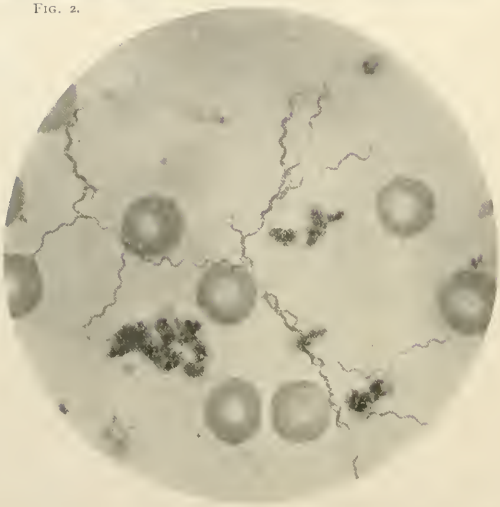

FIG. 3.

FIG. 4.

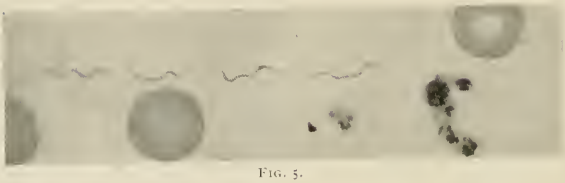

FIG. 5.

FOUR CASES OF GLANDERS IN MAN.

By Alwin M. Pappenheimer, M.D., *Pathologist.*
(From the Pathological Laboratory of Bellevue Hospital.)

The following report includes the detailed clinical and pathological records of four cases of glanders.* Without entering into a discussion of the literature, it may be justly said that the descriptions of the disease, as presented in the best-known American text-books of medicine and surgery, are utterly inadequate, and fail to give even an approximately accurate picture of the acute septico-pyæmic types of the disease. A study of the three fatal cases here described, each differing in many respects from the text-book descriptions, may therefore be of some value.

Case I.—H. M., stableman, age 55, admitted to service of Dr. William C. Lusk, April 4, 1905. Patient was delirious when admitted. No history regarding the details of his infection was obtained then or subsequently.

Physical condition on admission was as follows: Well-developed and well-nourished; skin hot and dry; old scars on forehead; ecchymoses on right eye; scar on lower lip; capillary ectasiae over both cheeks and nose; visible pulsation over vessels of neck; spastic condition of right arm and forearm; lymph glands not palpable; mucous membranes pale; old pigmented areas on both legs; right arm and shoulder greatly swollen and reddened, oedematous and very tender; patient delirious; tongue dry, heavy brownish coating, tremulous; pulse rapid, irregular; heart, lungs, abdomen, liver, spleen and reflexes, negative.

Patient after admission became rapidly worse. Because of a suspicion of erysipelas, he was transferred to the isolating pavilion, where the following note was made:

Skin over the right shoulder, extending down on arms, is of a crimson color, swollen and shining; shows slight oedema. On the top of the scalp there is a small wound. Skin extending outward in all directions from it is swollen, red, indurated and oedematous. Edges are undermined and separated from the healthy tissue by a sharp edge. Ecchymosis has appeared underneath the left eye. Mental condition: Patient is delirious, apathetic, and somewhat restless.

On the following day a pustular eruption developed over the body. Patient passed into coma, and died five days after admission. The temperature ranged between 102° and 105°, the pulse from 110 to 120, respiration between 30 and 40.

* Cases I. and II. were presented at a meeting of the New York Pathological Society; see Proc. N. Y. Path. Soc., Vol. V., p. 9, 173.

Urine—Amber; clear; acid; 1.020; albumen, trace. sugar, none; occasional granular and hyaline cast; few leucocytes.

Blood Count—On the day after his admission showed a leucocyte count of 10,000, of which 80 per cent. were polymorphonuclear neutrophiles; 5 per cent. large mononuclears; 15 per cent. lymphocytes.

A few hours before death, a blood culture was made. After 48 hours were seen on the blood-agar plate numerous whitish, viscid colonies, about 100 to a plate, which proved to be small Gram negative, irregularly staining rods, with the characteristic morphology of the bacillus mallei. Smears and streak plates on glycerine-agar were also made from a pustule on the forehead. These also showed the glanders bacillus.

An emulsion of a twenty-four glycerine-agar transplant from the blood culture, injected into a guinea pig, produced, after two days, the characteristic inflammations of the testes.

The autopsy was performed a few hours after death by Dr. Schultze, to whom I am indebted for the following report:

Scalp, over parietal and anterior occipital region, shows central defect, covered with dried scab. Around this, is an area four inches across, with numerous prominent, yellow foci, 1-3 mm. in size.

Over forehead are noted elevations, 1 mm. to 1 cm. in diameter, some with yellow head, and others open over the dome of the prominence. One on right eyelid, one on left chest, several on neck and supraclavicular region. A large one, two inches in diameter, over lower end of sternum, fluctuating at centre, yellowish, surrounded by red zone. On incision, brownish, thick pus (taken on swab).

On sides of abdomen, small nodules. Right forearm, abscess in muscle. On inner surface of right thigh, also nodule of same type as in forearm.

Diaphragm, third space right, fourth space left. Under area of abscess described on lower end of sternum bone shows yellowish infiltration of cancellous tissue; mediastinum shows quantity of yellowish-brown pus, with small surrounding foci.

Head—On section of the scalp, through area above described, the foci of suppuration are seen to extend down to the pericranium. The cellular tissue of the scalp, which is the seat of multiple abscesses, is oedematous. Over the frontal bones, just superficial to the pericranium, are several groups of small, yellowish foci, each surrounded by a hyperaemic zone. Calvarium normal.

Heart—Left ventricle contracted; right distended; blood mostly fluid; a few ecchymoses on pericardium, especially over left ventricle; valves are normal; muscle dark red, weight 12 oz.; aorta shows moderate degree of atheroma.

Lungs—Right lung: Apex adherent by old adhesions. On section are seen numerous foci, up to 3 cm., which on their pleural surface show yellowish centre, with numerous smaller yellow foci surrounding them, bounded by a hyperaemic zone. Left lung presents same lesions.

Tonsils enlarged to 2-3 cm.; oesophagus normal; on posterior wall of pharynx, on ary-epiglottic fold, in pyriform fossae, on epiglottis—

farcy buds up to 6 mm. in diameter; tracheal and bronchial mucosae normal.

Spleen—Weight 12 oz.; capsule shows numerous small areas of thickening; organ is firm; pulp is sufficiently soft to be slightly prominent.

Pancreas—Firm; white and opaque.

Stomach—Gastric mucosa is ecchymotic.

Liver—Weight, six lbs.; surface is mottled; vessels very distinct; intervening areas are of a light gray tint. A small adenoma, 3 mm. in diameter, on anterior surface; gall-bladder distended.

Adenals—Normal.

Kidneys—Left capsule free; surface smooth; cortex swollen and opaque; weight 6 oz. Right kidney 6 oz.; same appearence.

Intestines—Anatomical anomaly in relations of small intestine, mesentery, and descending colon.

Mesentery of small intestine attached along the usual position, but instead of presenting to the inner side of the ascending colon, it presents to the other side, so that the ascending colon runs from coecum, which has an unusually long mesentery, directly in front of spinal column, from fourth lumbar up to lower margin of stomach, then goes into hepatic flexure, where it goes directly under and to the inner side of the gall-bladder.

The duodenum curves in the shape of the letter "S," all on the right side of the spinal column, so that junction of duodenum and jejunum comes to lie on right side of second lumbar vertebra.

Intestinal mucosa congested. Small intestine contains fluid. Large intestine, fluid, with small amount of feces.

Brain—Dura not adherent; pia moderately oedematous, showing numerous grayish opacities, along the course of the vessels, over the sulci. On section, the brain is normal, save for a few small cysts in the choroid plexus in right lateral ventricle.

Anatomical Diagnosis—Glanders.

Bacteriological Examination—At the autopsy, streak plates were made from the heart blood, and the pus about the shoulder joint. From these, the b. mallei was recovered practically in pure culture. Typical bacilli were also found in smears from the pus.

Microscopic Findings—Sections were made from various deep and superficial pustules, tonsils, lung, liver, spleen and kidneys. The tissue was hardened in Muller-formol, imbedded in celloidin, and sections stained with iron-haematoxylin-eosin. The glanders bacilli were demonstrated in the abscesses with great difficulty. Stirling's gentian violet was the only successful stain; a number of others were tried, but did not show the bacilli.

Skin Lesions—The smallest nodules appear under the low power as more or less circumscribed areas of necrosis and cellular infiltration,

situated in the deeper layers of the corium, and not involving the epidermis. Under high magnification, they are seen to be composed of a mass of indefinite, granular necrotic material, in which lie numerous pycnotic nuclei, and chromatin fragments. These latter assume the most varied shapes, granular, globular, pyriform or thread-like. Frequently they appear to fuse into larger irregular, densely staining masses of nuclear material.

At the periphery of the abscesses are seen moderate numbers of apparently normal polynuclears and small round cells, together with leucocytes whose nuclei have already begun to show pycnotic and karyorrhetic changes. There are also a few large phagocytes. The margins of the abscesses are sharply demarcated from the surrounding tissue. The latter shows no proliferation of the fixed connective tissue elements, which might be interpreted as an effort at repair or encapsulation. The capilliaries, however, are intensely engorged with red blood globules, and there are numerous small haemorrhagic extravasations—the whole forming a zone of hyperaemia, completely surrounding the abscess. Swelling or proliferation of the capillary endothelium is not pronounced, nor do the blood vessels appear to contain an excessive number of lucocytes.

In the larger nodules the process has reached the epidermis, causing marked lesions of this tissue. The disintegration of the epidermis, however, takes place only over the summit of the nodules, which extend a considerable distance laterally beneath the epidermis.

The cells of the Malpighian layer first become swollen and hydropic; their nuclei are shrunken, no longer show nucleolus or chromatin network, and take a diffuse pale stain.

As they are torn apart, they may be seen lying upon the surface of the necrotic mass, faintly stained, but still recognizable.

At the margin of the pustule, in places, the cells of the Malpighian layer coalesce to form cysts of blebs containing irregular masses of a pale hyaline material, with a few polymorphonuclears and red blood cells.

The horny layer appears resistent to the advance of the necrotic process. In the smaller pustule, it forms an intact sheet over the surface of the abscess; in the larger pustules, it is broken up, and lies in shreds upon the surface of the exudate.

Abscesses of Deltoid Muscle—The section shows a number of irregular foci of suppuration, extending between the muscle fibres. These areas of suppuration and necrosis show essentially the same picture as the cutaneous lesions, save that, lying in the cellular detritus one may recognize fragmented and necrotic muscle fibres. There is extensive haemorrhagic extravasation in and about the abscesses. The capillaries are crowded with red globules, and the capillary endothelium appears swollen and proliferated. The endothelium of the lymph spaces likewise shows proliferative changes. The muscle fibers throughout the section show marked degenerative changes. The striations are indistinct or wholly lost. The fibres are greatly swollen, and on cross-section, have lost their fibrillar structure. There are large areas of partial necrosis, which stain poorly. The nuclei are swollen, pale and stain diffusely.

Tonsils—The normal lymphoid tissue is almost completely replaced by a mass of necrotic, purulent material, having the same character as that in the skin lesions. This necrotic process extends to within a short distance of the epithelium, but does not involve it. The intervening tissue appears oedematous, and the capillaries congested. The lymphoid follicles, left intact by the necrotic process, show evidences of active proliferation, their germinal centres being very marked.

There is no infiltration of the peritonsilar tissue.

Lungs—The pleura is thickened and fibrous. The pleural endothelium is swollen, cuboidal in shape, several layers deep. There is a slight amount of fibrinous exudate upon the surface, and a few exfoliated cells and polymorphonuclears.

The section shows also disseminated abscesses, having the same structural features as in other tissues. There is no trace of remaining pulmonary tissue in these areas; they shade, however, into areas of lobular pneumonia, where the alveolar structure is well preserved. The air spaces here are more or less filled with non-fragmented polymorphonuclears, red globules, fibrin in small amount, and a few exfoliated pigmented alveolar cells. The alveolar capallaries show marked engorgement. In other places, the pulmonary tissue is practically normal in appearance. The bronchi, save in the regions affected by the necrotic processes, are normal. A large vessel, surrounded by healthy pulmonary tissue, contains a red thrombus, one end of which is formed by a mass of broken-down nuclear detritus, identical in appearance with the contents of the abscesses.

Liver—The vessels in the perilobular spaces, as well as the liver capillaries, are filled with red globules. The parenchymal cells appear somewhat swollen and hazy. There are no other noteworthy changes.

Spleen—Shows marked congestion.

Kidney—Shows acute parenchymatous degeneration of moderate grade.

Case II.—B. A., age 35; stableman; admitted on December 1 to the service of Dr. Alexander Lambert, who has kindly placed the clinical facts at my disposal.

Family and previous history of no bearing on his present illness. Patient is a stableman in a coal yard, Avenue A and Nineteenth street. Six weeks ago was bitten in the thigh by a horse. The wound healed rapidly and caused no further trouble. The horse which bit him was apparently healthy at the time and remained so. About four weeks ago the man for whom he worked bought a horse which became ill a few days later and developed typical farcy nodules. At the end of a week this horse was shot by the order of the Board of Health. During this week patient groomed and fed the horse and probably became infected either by inhalation or through the wound in his thigh, which had at this time not entirely healed. Three days before entering the hospital he went to work feeling as usual, but during the day noticed a sharp pain in his left side. A little later he had a rather severe chill. The following day, high

fever, cough, with scant whitish expectoration and some dyspnoea. Pain in the side was persistent and severe.

Physical Examination—Showed a well-developed male. Rapid grunting expiration; skin hot; appeared acutely ill.

Lungs—Over the left lower lobe, dullness, absent fremitus; diminished breath sounds, and many coarse crepitant rales. Physical examination in other respects was negative. The urine was of high specific gravity and contained a considerable amount of sugar.

During the following twelve days the temperature continued high, ranging between 102 and 104.5, resembling typoid or an acute general miliary tuberculosis. The patient rapidly became profoundly septic, tongue dry, pulse progressively more rapid and weaker. Respirations kept about 40, and his general condition grew daily worse. His appearance suggested profound sepsis. The blood examination showed a very slight leucocytosis, 13,000 to 15,000, save just before death, when there was an agonal rise to 19,500. Widal reaction on December 30 was negative. No cutaneous eruption appeared at any time during his illness, but about three days before his death the House Physician first noted a number of deep-seated, nodular swellings, scattered over the trunk and extremities. The skin over these showed inflammatory signs, and indistinct sense of fluctuation was obtained over the larger ones. About the same time, there was noted also over the third meta carpo phalangeal joint of the right hand, and over the right knee joint, swelling, redness, heat and marked tenderness.

On December 31, two days before death, blood culture was made. At the end of 48 hours, the blood agar plates showed numerous deep and superficial colonies, about 35 to a plate, the superficial ones being raised, whitish, opaque and viscid. The flasks showed a flocculent sediment, which on examination showed a small, slightly bent, irregularly stained Gram negative bacillus, strongly suggesting B. mallei. Subcultures on potato showed, after a few days, a characteristic, raised viscid brownish growth.

Post-mortem examination, performed ten hours after death, showed the following: Poorly nourished male; apparent age, 35; medium frame; small superficial abrasion, covered by scab on external surface of right thigh. Over both arms, on dorsum of right hand, over left thigh on external surface, and scattered irregularly over the rest of the body, are a number of deep-seated, fluctuating nodular swellings, showing as diffuse rounded prominences of the skin, without external signs of inflammation. Section into these shows them to correspond to deep abscesses localized chiefly within the muscles and deeper layers of the corium. Abscesses contain a thick, yellowish-white or brownish pus. There is apparently entire absence of inflammatory reaction about them; neither granulation tissue nor hyperaemia. The muscles are firm and reddish brown in color. Right knee slightly swollen, and on section exudes thin, purulent fluid. There is swelling over third right metacarpophalangeal joint; incision followed by escape of thin pus. Peritoneal cavity and appendix normal. Left lung is adherent over the lower half of the lower lobe, to the chest wall. Right lung is free.

Heart—Pericardium is normal. Heart rather small. Myocardium brownish red in color. Valves and orifices normal.

Lungs—Left lung: over the lower portion of the lower lobe, the pleura is thickened, showing patches of firmly adherent fibrin, and superficial circular yellowish abscesses. (Abscesses of the pleura?) Bronchial lymph nodes are small, anthracotic, and the seat of calcareous deposits. Bronchi contains frothy fluid. Otherwise normal. Parenchyma of the lung, save for moderate oedema, normal. Right lung shows moderate oedema; otherwise normal.

Liver—Normal in size and consistency. Color reddish-brown. Markings fairly well defined. Gall-bladder normal.

Spleen—Slightly enlarged; capsule tense. On section it is somewhat softer than normal; pale in color. No undue prominence of Malpighian bodies or trabeculae.

Pancreas—Pale and firm.

Adrenals—Normal.

Kidneys—Normal size. Capsule strips easily. Surface is smooth. Cortex of normal width. Markings indistinct. Color reddish brown. No gross pathological changes.

Bladder and Ureters—Normal.

Pharynx—Normal.

Larynx—Left arytenoepiglottic fold and the mucous membrane in the pyriform sinus on the left side is markedly oedematous. There is a fine erosion of the left cord. The anterior wall of the larynx, just below the rima glottidis, shows a group of small submucous abscesses, which exude thick, yellowish-white pus. These show no surrounding inflammatory reaction. Mucous membrane of the trachea elsewhere is normal.

Stomach—Shows numerous ecchymoses. Slight swelling of the mucous membrane. In the pyloric half the mucous membrane is mammillonated. This condition is marked also in the first portion of the duodenum.

Small and Large Intestines—Normal.

Thyroid—Normal.

Brain—Calvarium is normal. Dura is not adherent. Vessels of the pia arachnoid are moderately injected, and the sub-pial fluid is slightly increased. The lateral ventricles contain only a few c.c. of clear fluid. The telae are oedematous. Ependyma is smooth. Brain substance is firm, and shows no pathologic change, except engorgement of the capillaries.

Anatomical Diagnosis—Pyaemia, glanders, abscesses of muscles, pleurae, laryngeal mucous membranes. Smears were made from the laryngeal abscesses, and from several of the subcutaneous lesions. Cultures were made from the spleen and from the pus in a deep nodule in the right arm. A guinea pig was injected intraperitoneally, with a broth suspension of pus from a subcutaneous abscess. The B. mallei was obtained in pure culture from the spleen and abscess, and bacilli having

characteristic morphology and staining reaction were seen in smears from the pus.

The guinea pig showed after 24 hours, slight swelling of the testicles, which, at the end of 72 hours, was very marked, the overlying skin being hot and glazed. Three days after injection, the pig was killed. Autopsy showed superficial abscesses in the seminal vesicles, and intense fibrino-purulent inflammation of the tunica vaginalis, which was studded with small miliary abscesses. The testes proper were swollen to about twice their normal size. There was no peritonitis. Pure cultures of the B. mallei were obtained in streak plates on glycerine-agar from the peritoneal cavity, and the tunica vaginalis.

Microscopical Examination—Tissue was taken for examination from abscesses in muscles about elbow, pleura and lung, myocardium, liver, spleen, kidney and pancreas. Fixation in Müller-formol, imbedding in celloidin, staining with iron haematoxylin-eosin.

Section Through Deep Abscess About Right Arm—The section shows irregular areas of necrosis, cellular infiltration between the muscle fibres, and foci of suppuration. In the necrotic areas one may distinguish pale, thick bands of fibrillar structure, probably representing necrotic muscle fibres and a granular deposit of fibrin. There is also considerable haemorrhagic extravasation, and moderate infiltration, with plasma cells, polynuclears, and a few larger epitheloid cells.

Here and there are dense collections of cellular elements, evidently abscesses consisting of polymorphonuclears and fragmented chromatin particles, lying in a necrotic matrix. These are very similar in appearance to the abscesses seen in Case I.; nevertheless, the lytic changes in the nuclei are less extreme, and there are not to be seen the large distorted masses of chromatin which were a striking feature in the first case.

These abscesses, though irregular in distribution and extent, are usually sharply circumscribed. In places, however, the adjacent inter-muscular septa are infiltrated with leucocytes. There appears to be considerable swelling and proliferation of the endothelium of the lymph-spaces, with the formation of epitheloid cells, showing large, pale vesicular nuclei.

The muscle fibres appear to necrose en masse. Adjacent to areas where the necrotic process is extreme and no trace of normal tissue persists, one may see individual muscle fibres, which preserve their striations and show little change from the normal. In general, the fibres are somewhat swollen, stain poorly with eosin, and show swelling and proliferation of their nuclei.

Sections from Lung and Pleura—The pleura shows considerable fibrous thickening, oedema, intense congestion, and moderate pigmentation. The section passes through two flat nodular masses of exudate, situated upon the surface of the pleura. These consist entirely of small round cells, red globules, a few polymorphonuclears, and indefinite granular detritus. There is no fragmented nuclear material, such as was seen in the first case. Beneath the pleura, in the vicinity of the nodules, the lung shows small areas of lobular pneumonia of the usual type. The alveoli contain masses of polymorphonuclears, red globules, and

fibrin. The bronchi show slight exfoliation of the epithelium, their lumen is filled with polymorphonuclear leucocytes.

Liver—Slight albuminous degeneration of the parenchymal cells, and capillary congestion.

Spleen—Splenic follicles are small and show no germinal centres. Pulp cells are increased. Trabeculae normal. No congestion.

Kidney—Congestion and actue parenchymatous degeneration.

Pancreas—No noteworthy change.

Myocardium—The muscle fibres show fragmentation. The striations are very indistinct. The nuclei are swollen and pale. The intermuscular capillaries are congested.

Case III.—J. S., age 24; male; driver; born in United States; admitted to the Fourth Medical Division, service of Dr. Nammack, on November 30, 1906.

Family History—Father died of tuberculosis. Otherwise negative.

Personal History—Alcohol: averages one glass of whiskey and four or five glasses of beer daily; frequent periodical excesses. No venereal diseases. Measles and whooping cough in childhood. Fracture of left leg when five years old.

Previous illnesses—Patient's health was excellent until the previous winter. He then acquired a cough, with muco-purulent sputum, and was ill for five months (weeks?) at the Metropolitan Hospital in the tuberculosis ward. He improved greatly, but has had a slight cough ever since. He has never had hemoptysis.

Seven months ago he had an abscess over the left temporal region. This healed (?) but broke out afresh four weeks ago.

Present Illness—About eleven days (two weeks?) ago, the patient was taken ill with headache, fever, slight cough and general prostration. About the same time, he had considerable pain in his left leg at the site of the old fracture; this disappeared after a few days. His general symptoms, however, continued, and to them were added anorexia, diarrhoea and frequent vomiting. He has been in bed most of the time.

There has been no chill or epistaxis.

Chief Complaints—Weakness, fever and diarrhoea.

Physical Examination upon Admission: General Appearance—Well-built, well-nourished male; face flushed; apathetic; does not appear acutely ill. Abscess and small cicatrix of previous incision in left temporal region.

Eyes—Pupils normal in appearance and reactions.

Tongue—Dry, large. Brown coat on dorsum. No tremor; no deviation.

Heart—Left border, 3½ inches to left of mid-sternal line. Right border at right sternal margin. Action regular and of good quality. Sounds clear; no murmurs or accentuations.

Lungs—Resonance and vesicular murmur good.

Abdomen—Soft, rounded, not tender. No masses or fluid. A small number of roseolar spots, very indistinct, scattered over abdomen.

Liver—Edge just palpable below ribs.

Spleen—Edge felt ¾ inch below costal margin.

Reflexes—Patellae normal.

Temperature—102°; pulse 90; respirations 20.

Urinalyses—December 4: Clear, dark amber, acid; sp. gr. 1028, no albumen, no sugar, no casts. December 12: Turbid, yellow, acid; sp. gr. 1018, no albumen, no sugar, no casts.

Further Course of Illness—The *temperature* during the remainder of his illness ran a high continued course with remissions. For the first eleven days it ranged between 100° and 104°, the curve showing a tendency toward an evening rise. From this time, until his death, it remained at a still higher level, ranging from 101° to 105°. During his last twenty-four hours the temperature twice reached 105.6°. The *pulse* gradually became more rapid and of poorer force. Toward the end the pulse rate varied between 120 and 150. The *respiratory rate* varied with the temperature, but was relatively slow, ranging between 24 and 28. Immediately before death the respiration became very rapid (68).

The patient remained in much the same condition as upon admission until December 11, when it is noted that he complained of pain in his left leg. On the following day his right knee also became painful. On December 17, five days later, a note by the house physician describes the right knee as "swollen, tender, but not reddened. The enlargement is most marked about the patella and its synovial extensions." Two days later, the patient became restless and delirious; the delirium continued, passing gradually into deep stupor. The right knee became more swollen, tender and inflamed. A prepatellar incision was made on December 24; the pus was not examined bacteriologically.

Blood Examinations—December 4: Leucocytes, 4,900; differential count, polymorphonuclears = 74.5; lymphocytes = 23.5; mast cells = 1.0; eosinophiles = 0.5; transitional's = 0.5; morphology of red cells normal. December 10: Leucocytes = 6,000. December 23: Leucocytes = 8,800.

Widal Reaction—December 4, negative.

December 6, negative.

December 26, negative.

Blood Cultures—December 8, sterile; December 21, sterile.

The patient died on December 27. Total duration of illness, six weeks approximately.

Chief Symptoms—Continued fever, prostration, diarrhoea, arthritis of right knee. No leucocytosis; no cutaneous lesions.

Post-mortem Examination—Performed twenty-seven hours after death, showed the following: Tall man; moderate emaciation; pubic hair, female type; axillary hair, undeveloped; axillary lymph nodes barely palpable; inguinal lymph nodes about normal in size. Panniculus, ¼ inch; rigor mortis present; moderate pallor of the skin; no oedema; peritoneal cavity is normal, there being no fluid; transverse colon markedly distended with gas; peritoneal serosa slightly thickened; diaphragm fifth space on both sides; left pleura free from adhesions; no fluid.

Lungs—Slightly emphysematous; no barrel-shaped chest; lungs are well aerated; moderately anthracotic, with a few firm nodules in the upper lobe (healed tubercles). On section lung is slightly hyperaemic; no tubercles or areas of lobular pneumonia seen; bronchi longitudinal striations are prominent; mucous membrane slightly hyperaemic; bronchial lymph nodes small and anthracotic. Right pleura free from adhesions and fluid; right lung, firm nodules at the apex, small pea size. The nodules that are present in the upper lobe are reddish in color, sharply circumscribed and granular; lower lobe somewhat firm and hyperaemic; hypostatic congestion. In the middle lobe there is a small area about 1 cm. in diameter, which is sharply circumscribed, and on section reddish, and in places partly softened.

Heart—Pericardial cavity normal; no fluid; over the right auricle the pericardium is slightly roughened; over the region of the thymus tissue is present, which resembles thymus tissue on cross section; right lobe of the thymus forms a long tongue-like projection, being absent on the left side. Right heart and its valves normal; heart muscle decidedly pale in color; aortic valves are normal, and show slight fenestration; mitral valve normal; aorta absolutely normal; likewise coronary; heart muscle, though pale, is firm; endocardium of left ventricle slightly opaque.

Spleen—Weighs 365 grammes; adhesions between the diaphragm and the adjacent viscera. Spleen is firm; on section dark red; splenic follicles in places are enlarged to the size of small pin-heads. Mesenteric lymph nodes are small, and have a decided yellowish appearance; lacteal vessels in places are enlarged.

Liver—Weight 2.260 grammes; large; on cross section marked perilobular fat infiltration. In the right lobe there is a large triangular area with base on the superior surface, reddish in color. Examination of the portal veins shows no thrombosis; portal vessels, bile-ducts, etc., appear normal, as well as the lymph nodes; gall-bladder contains a small amount of yellowish bile; no stones; otherwise normal.

Larynx, Mouth and Tonsils—Normal.

Oesophagus, Stomach, Small and Large Intestines—Normal, except for a few areas of slight congestion, and distension of the blood vessels in the small intestines and in the jejunum and large intestine.

Over the right knee joint there is a surgical incision. Subcutaneous tissues are markedly thickened and infiltrated with pus. Several small blebs of skin, 1 cm. in diameter, filled with thick reddish pus, found in lower region of the knee over tibia. Joint cavity somewhat swollen, and on opening, a yellowish fluid, containing floccules of pus, exudes.

In the left temporal region, there is a large, irregular superficial ulcer, with the floor formed by clean granulations. Beneath this, on removing scalp, no necrotic bone is found.

Brain—Dura not adherent; pia normal; no congestion of the cerebral veins; lateral ventricles are not distended; no fluid at the base; vessels at the base normal; on cross section, brain appears normal. Ears and sphenoids normal.

Suprarenals—Normal in size and appearance.

Kidneys—Weight 440 grammes, together; somewhat large and firm; capsule non-adherent; cortex enlarged, very pale; pyramids congested. One of the pyramids of the left kidney contains an area of fibrosis (fibroma).

Bladder—Markedly distended.

Prostate—Normal.

Thyroid—Normal.

Anatomical Diagnosis—Glanders (?).

Status Lymphaticus (slight).

At the autopsy the possibility of the case being one of glanders was entertained, although a final diagnosis was reserved; pending the outcome of the bacteriological examinations. The points suggesting glanders were (1) the occupation of the patient; (2) the peculiar nodular abscesses in the lungs, and (3) the suppurative arthritis and cutaneous pustules about the right knee.

Characteristic intramuscular abscesses or "farcy buds" upon the respiratory mucous membrane were not present.

Bacteriological Examination—Smears made from pustule about right knee joint showed fairly numerous delicate, irregularly staining, slightly curved bacilli, decolorized by Gram's stain. Cultures from the pustules, from the purulent exudate in the knee joint, and from the spleen, proved unsuccessful. The plates from about the knee, and from the joint fluid were overgrown with contaminating organisms; those from the spleen were sterile. Pus from the knee joint and from the subcutaneous abscesses was collected on swabs, and two male guinea pigs inoculated intraperitoneally with broth suspensions. A third pig was inoculated with a broth suspension of a nodule in the lung. Of these, all developed as intense orchitis, noticeable even after 24 hours, and showed the signs of an intense infection. Pig No. 1, inoculated with pus from the knee joint, was killed on the third day; the testicles were greatly swollen, and there was fibrinous deposit and the formation of miliary abscesses upon the tunica vaginalis and peritoneum. Smears from the exudate showed great numbers of Gram negative rods, morphologically typical of B. mallei. A pure culture of the glanders bacillus, showing characteristic cultural features, was readily obtained. Pigs No. 2 and No. 3 died spontaneously on the third and fourth day, showing the virulence of the strain. Autopsy showed intense and characteristic lesions.

Microscopical Examination: Skin about knee joint—The section shows a number of subcutaneous abscesses, some of which are fairly circumscribed; in other places, there is a diffuse purulent infiltration, which extends upward into the corium, and infiltrates the papillae. The epidermis in places is raised from the corium, forming blebs filled with purulent exudate; the process, however, has originated in the subcutaneous areolar tissue.

The abscesses consist of dense aggregations of polynuclear leucocytes. In some places there have undergone karyolytic changes, and the field is filled with globules and irregular fragments of nuclear material. There is capillary extravasation and oedema of the adjacent cellular tissue.

The larger blood vessels are engorged with blood; some of them contain suppurative thrombi. About others there are collections of lymphoid cells. Some of the smaller, deep-seated abscesses show a more or less definite capsular arrangement of the adjacent connective tissue, indicative of an attempt at repair.

Lungs—The nodular foci noted in the gross prove to be sharply circumscribed abscesses having the same general characters as those described in the previous cases. There is marked proliferation of the interstitial tissue at the periphery of the abscesses, and intense engorgement of the alveolar capillaries.

Thymus—A few small islands of lymphoid tissue, with indistinct Hassall bodies, are found.

Myocardium—The intermuscular spaces appear oedematous, and the striations of the muscle fires are cloudy and indistinct.

Pancreas—Slight interstitial pancreatitis.

Spleen—Marked congestion, with areas of haemorrhage. The Malpighian follicles are small.

Kidneys—Intense congestion; slight cloudy swelling of the epithelium of the convoluted tubules. Fibroma of one pyramid.

Other Organs examined show no noteworthy lesions.

These three cases are of considerable interest clinically, as illustrating the acute septicemic type of the disease. The total duration of the illness in Case I. is undetermined; in Case II., 33 days; in Case III., approximately six weeks. Two of the patients were stablemen, the third a driver, so that in each case direct infection from infected horses may be assumed. In Case II., in which, after the diagnosis had been established, a careful inquiry was made, the incubation period was determined quite definitely as being between three and four weeks. This is considerably longer than the period given in the text books (4 to 6 days—Leube).

The mode of onset in Case I. is unknown; in Case II. it strongly resembled that of lobar pneumonia, the patient being suddenly stricken with a chill, high fever, cough, severe pleuritic pain and dyspnoea; in Case III., the mode of onset resembled that of typhoid fever or acute miliary tuberculosis—prostration, headache, nausea, diarrhoea and cough.

The general symptoms in all the cases were strikingly similar. The temperature was continuously high, with daily remissions of two or three degrees. There was intense prostration, restlessness, ending in delirium and stupor. In Case III., vomiting was present, and diarrhoea a prominent symptom early in the disease.

The *cutaneous lesions* varied in each case. In Case I., there were subcutaneous abscesses of the scalp and about the right shoulder, and on the day before death a general pustular eruption. There were also noted ecchymoses about each eye. In Case II. there were no cutaneous lesions, properly speaking. Three days before death there were observed for the first time scattered nodular, deep-seated swellings over the trunk and extremities, none of them reaching the surface at the time of death.

In Case III. there is noted an "indistinct roseola" over the abdomen, probably rendered more distinct by the strong suspicion of typhoid fever which was then entertained. No further notes of any cutaneous lesion are recorded, but at the autopsy there were found a few pustules and deeper abscesses about the right knee. These may, however, have been due to a secondary infection of the skin and subcutaneous tissue following incision into the knee joint, and evacuation of the infected joint fluid. No other skin lesions were present.

Suppurative arthritis occurred in each case. In Case I. the right shoulder was involved; in Case II. the right knee and one of the metacarpo-phalangeal joints; in Case III. the right knee joint.

The lungs and pleura were affected in all three cases. In Case II., however, the respiratory symptoms dominated the clinical picture in the early stages of the illness, and were doubtless referable to the intense, localized pleuritis, found at autopsy.

The blood picture was not characteristic. Case I. showed a slight leucocytosis (10,000), with an increasey polynuclear count (80%), and absence of eosinophiles; in Case II. there was a moderate leucocytosis (13,000, 15,000, 19,500); in Case III. the total count at no time exceeded 9,000, and the differential count was within normal limits.

The glanders bacillus was readily isolated from the blood from Case I. a few hours before death; in Case II. two days before death. In Case III. blood cultures were taken nineteen days and six days before death, but were unsuccessful. Unfortunately only fluid media (broth flasks) were used, and it is possible that a more careful technique would have demonstrated the organisms.

In Case I. the diagnosis was made before death by Dr. Norris, from the appearance of the cutaneous lesions and abscesses. In Case II. the diagnosis of glanders was suggested by the house physician, Dr. Pilcher, and confirmed by blood culture. In Case

III. the diagnosis remained obscure, until a bacteriological examination of the pustules and joint fluid at autopsy conclusively proved the nature of the illness.

I am greatly indebted to Drs. Nammack, Lambert and Lusk for permission to utilize the clinical records, and to Dr. Norris for the autopsy report, assistance in the bacteriogical examinations and in the preparation of this paper.

Through the courtesy of Drs. Bryant and Cramp, we are able to add the brief report of a fourth case of infection with the glanders bacillus. This case will be presented in detail by Dr. Cramp in a later publication.

Case IV.—The patient, M. M., male, 34 years of age, a laborer in the Street Cleaning Department, was admitted to the service of Dr. Bryant on October 20. Four weeks previously he was taken ill with chills and fever, which recurred several times a week up to the present. Three weeks ago there developed an abscess of the right leg, and recently similar abscesses have appeared on the forearms.

Examination showed a number of deep-seated areas of fluctuation, without surrounding induration or superficial heat or reddening, and but slight tenderness. During his stay in the hospital the abscesses were successively opened and drained, and healed up promptly. The temperature was of an irregular pyaemic type, ranging from normal to 102.6. There was little prostration, and the constitutional disturbances were slight. A blood count taken on October 21 showed 11,400 leucocytes, with a normal differential count.

The patient was discharged on November 5 "cured," save for a slight discharge from the incision of the large abscess in the right thigh. The patient has since been under observation; so far as can be ascertained, he is now completely recovered of his illness.

Blood culture taken the day following admission was sterile.

Pathological Report—A deep abscess, situated on the posterior and outer aspects of the middle third of the right thigh, was aspirated with a sterile syringe, about 5 cc. of pus being withdrawn.

Morphological examination of this pus on smears and in hanging drop revealed no micro-organisms.

Cultures made upon slanted and coagulated Loeffler's blood serum, and on agar tubes, remained sterile after a week in the incubating oven. With the pus two guinea pigs were inoculated, one subcutaneously, the other intraperitoneally.

On November 7, seventeen days after inoculation, the subcutaneous pig was found greatly emaciated, and had developed at the site of inoculation an abscess of considerable size, say 3 cm. in diameter. The abscess was incised, and the pus evacuated. Smears made from this pus revealed a few Gram negative rods, which closely resembled in their morphology the B. mallei. Besides these rods of distinctive morphology, there was found a number of involution and degeneration forms, and of globules and

round masses, not staining by Gram's method, but taking on an after stain with fuchsin.

Cultures made from this pus yielded in forty-eight hours, upon slant glycerin-agar, a growth of Gram negative rods, resembling the glanders bacilli in morphology. An emulsion of glycerin-agar slant was made and inoculated into two large male guinea pigs, each pig receiving approximately half of the agar culture emulsion.

The pigs showed a marked and prompt reaction after inoculation, and the morning following the inoculation, both pigs were very ill, with hair standing on end, extremely sensitive to the touch, especially on the peritoneum, and with greatly enlarged testicles. The pigs were found dead the following morning, approximately thirty-six hours after inoculation.

Examination of both pigs showed a diffuse general sero-fibrinous peritonitis, and the typical exudate upon the tunica vaginalis. The organism was found in the exudate of both guinea pigs in smears, and by culture. The two guinea pigs inoculated with the human pus were greatly emaciated, and were chloroformed on November 13, in a moribund condition, twenty-three days after inoculation. The subcutaneous pig showed no lesions in the gross beyond the abscess at the site of inoculation in the skin. The intraperitoneal pig showed a number of well-encapsulated abscesses in the peritoneum and retroperitoneal lymph-nodes, and in the skin at the site of the puncture of the needle, and abscesses in the liver.

The bacillus which was isolated corresponded in all morphological characteristics with that of the B. mallei. It decolorized by Gram's stain. The cultural characteristics corresponded in every way to that of the glanders bacillus, giving a characteristic growth upon potato and blood serum.

Microscopic examination of the tissues of the lesions of the two guinea pigs inoculated with human pus show the typical lesions of glanders, with numerous characteristic bacilli.

SOME GENERAL CONSIDERATIONS ON THE PNEUMOCOCCUS AND PNEUMOCOCCUS INFECTIONS.*

By Alwin M. Pappenheimer, M. D., *Pathologist.*

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It was with a good deal of hesitancy that I accepted Dr. Coleman's invitation to discuss before you the subject of Pneumococcus infections. Having learned to recognize in the pneumococcus one of the most versatile of pathogenic bacteria, which may cause anything from a conjunctivitis to a fulminating septicæmia, to treat of pneumococcus infections with any degree of thoroughness, seemed to imply covering the whole range of medical practice. It would have been easy to inflict upon you a long list of lesions, in which the pneumococcus may be concerned, and to exhaust my allotted time in so doing.

Rather than undertake such a distressing task I have thought it better to discuss briefly certain definite phases of the subject, and rather from the laboratory viewpoint than from the clinical. If what I say appears somewhat desultory, I must ask for indulgence on the ground that my subject is far too comprehensive to compress into twenty minutes' talk.

I wish first to speak of the difficulty in the identification of the pneumococcus—difficulties which are ignored in most clinical articles, and in many of the earlier purely bacteriological works It is a curious fact that the recent refinements in our bacteriological methods have merely succeeded in making more complex and difficult the precise differentiation of the pneumococcus from similar organisms.

The old criteria of identity have proven, one after the other, to be inconstant. Many atypical strains have been studied which more or less closely resemble the pneumococcus on the one hand and the streptococcus on the other. These intermediate types, moreover, have proven to be of practical importance to the bacteriologist. The precise significance from the clinical point of

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view it is perhaps too soon to estimate, but that they are frequently encountered and in a wide variety of pathological processes no one who has had any first-hand experience in routine bacteriological work can doubt. Thus, in a routine examination of lungs at autopsy during our work in the pneumonia commission, Dr. Norris and I[1] found such intermediate types present in 40 per cent. of our cases.

At present we have as our best criteria for the identification of the pneumococcus: First, the presence of capsules, more readily demonstrated than formerly, with the improved capsule stains of Professor Hiss[2] and Dr. Buerger[3]; second, the more or less distinctive greenish discoloration of the colonies on blood media; third, the specific fermentation of the complex sugar media; fourth, the customary virulence of the pneumococcus for mice and rabbits; fifth, the specific agglutination reactions. There are minor differential features, but none that approach in constancy or importance these five.

I have mentioned these differential points only to emphasize the fact that not one of them is always to be relied upon. There are pneumococci, orthodox in all other respects, which cannot be made by the most dexterous laboratory juggling to take upon themselves a capsule. Conversely, there are streptococcus-like organisms, not to mention that much discussed variety—the streptococcus mucosus—which have distinct and easily demonstrable capsules. Although in general the capsules of the streptococci and of the mucosus type differ in some respects from those of the typical pneumococcus, still in many individual instances the variations from the normal type are such as to make this guide to identity worthless.

The pneumococcus, as is well-known, produces upon blood-agar plates very characteristic greenish colonies, surrounded by a narrow zone of incomplete hemolysis. The streptococcus colony on the other hand is most commonly grayish, and is inclosed by a relatively broad transparent zone of hemolysis. There are, however, exceptions to this rule on both sides. Libman,[4] Ruediger[5] and others have described actively hemolysing pneumococci; and there is a rather frequently encountered type of streptococcus—the streptococcus mitior or viridans of Schottmueller—which produces a distinct greenish colony (plate). Other strains, again, produce a green color only upon the addition of glucose to the agar.

The inulin reaction is perhaps our best differential character between the pneumococcus and the streptococcus. Yet this, too, may prove uncertain. It is rare indeed to meet with pneumococcus-like organisms which do not ferment inulin more or less actively; on the other hand, it is a not infrequent occurrence to find varieties which in all other respects resemble streptococci, but which so far forget themselves as to ferment inulin. We have encountered three such strains, each of them highly vigorous inulin fermenters, but in no other way showing affinities with the pneumococcus.

That the animal reactions are not specific, and depend largely upon the virulence of the particular strain studied and the freshness of the isolation is well known.

It has been shown by Washbourne,[6] Wadsworth,[7] Rosenau[8] and others that if the pneumococcus be grown on blood media the virulence deteriorates very slowly, and may be maintained to a considerable degree for months. This precaution has been almost entirely neglected, and in consequence the deductions of most writers as to the virulence of the particular strain studied are quite untrustworthy. Moreover, it is not always safe to draw inferences as to the virulence for human diseases from the result of inoculations into mice or rabbits. Pneumococci living as inoffensive saprophytes in the normal mouth may be highly virulent for mice; vice-versa, a pneumococcus isolated from a case of fulminating septicæmia in man may leave a mouse unscathed. It is said by Ruediger and others that the intermediate diplococci which produce green colonies on blood agar and correspond to the diplococcus viridans of Schottmueller, have but a faint pathogenicity for rabbits and mice. I have recently isolated from the blood of an acutely fatal septicæmia, complicating erysipelas, an organism of this type which, even in the first transplant and in large doses, failed to kill white mice.

Finally, when we came to apply the agglutination test with specific immune sera we again meet with difficulties. The technical obstacles in obtaining uniform emulsions may be partly overcome by the use of Dr. Hiss'[9] calcium carbonate broth; but the agglutinative relations of the various strains appear to be inconstant, and repeated tests with the same strain often show unexplainable discrepancies.

If there is a moral to be drawn from this list of uncertainties it is this: The literature on pneumococcus infections of every sort

is enormous, yet in the greater number of reported clinical cases the identification of the inciting organism is not above suspicion. Of course, one cannot expect any type of micro-organism always to live up to its text-book attributes; in many instances the exact identification is of purely academic interest. On the other hand, it is possible that these varying types of diplococci may produce varying clinical pictures, and it may prove to be of practical importance to determine whether we are dealing with a straight pneumococcus or with a streptococcus mucosus or with one of the little-studied "intermediate" forms. So far we have little evidence that such is the case, and the few examples which I have seen of infection with "intermediate" organisms—cases of acute malignant endocarditis and meningitis—have not deviated strikingly in their clinical aspects from similar infections with the typical pneumococcus. Nevertheless, the question is one which has been insufficiently studied and deserves attention.

The question as to whether the pneumococcus is invariably present in the blood-stream at some time during the course of a lobar pneumonia—in a way comparable to the Bacillus typhosus in typhoid, has been much discussed, and is of considerable theoretical interest. The earlier investigators—Sittman,[10] Cohen,[11] Sello,[12] Kuhnau,[13] Nazari,[14] Fraenkel,[15] Beco[16] and others, working with blood agar plates, isolated the pneumococcus from the blood in from 20 to 30 per cent., the positive cases, as a rule, tending to end fatally. So also Lenhartz,[17] working over a large series of cases, but with solid media, succeeded in less than a third of the cases—and over a half of these were fatal. He concluded that the pneumo-coccemia occurred only where the bactericidal powers of the blood were in abeyance—usually before death. Schottmueller,[18] out of 209 cases of lobar pneumonia, succeeded in but 23 per cent., also using solid media. Although he states that he controlled his results in many instances by using bouillon as well as agar, he gives but one illustration of such a control experiment. In this he used 7 cc. of blood, dividing it among 6 agar plates and one broth tube. A few pneumococcus colonies developed on one of the plates—none in the single broth tube; obviously, with this unfair division of the blood, his negative result is not important in deciding the question as to the relative merits of solid and fluid media in the detection of the pneumococcus.

Among the observers who have achieved much better success in finding the organism in the blood are Baduel[19] (55 out of 57), Silvestrini and Sertoli[20] (15 out of 16), Bergheimi[21] (8 out of 8), Prochaska[22] (50 out of 50), and, finally, Rosenau[23] (132 out of 145 cases). These greatly improved results are attributed in all probability to the use of fluid media for cultural purposes—as in all other respects, the technique of the less successful workers is identical.

We must conclude, then, from the literature, that with suitable technique the pneumococcus may be demonstrated in the peripheral blood in practically every case of lobar pneumonia, and that its presence is in nowise indicative of a fatal termination.

Pneumococci have been found as early as the second day of the disease, and in several instances before the appearance of the physical signs. They persist, and may be found with considerable regularity until within twenty-four to thirty-six hours of the crisis when, in the majority of cases, they disappear from the blood. Occasionally they may be obtained after the critical defervescence, and very rarely they have been found for a week or more afterwards. In the fatal cases, especially those associated with hypo-leucocytosis, there is usually to be observed a pronounced ante-mortem increase in their number.

In connection with the subject of blood cultures there arises the interesting and much debated question as to the pneumococcidal action of normal or pneumonic blood. I have already stated that Lenhartz and the older investigators attributed their failure to grow pneumococci on blood-agar plates in many instances, to the supposed bactericidal property of the blood. This idea has been partially disproved, as regards the growth of the pneumococcus in vitro, by the high percentage of positive results obtained with very low dilutions of blood in broth media. Indeed, Rosenau, in his first article, states that the organism multiplies rapidly in pure defibrinated normal or pneumonic blood, as shown by actual numerical determinations. In a later and more careful study, however, he concludes that defibrinated pneumonic blood has a definite pneumococcidal action, definitely proportional to the leucocytes present, and gives evidence to show that it is due entirely to phagocytosis (at least in vitro). That this anti-pneumococcidal action is not produced by bacteriolytic substances in the serum is shown by the fact that pneumococci grow readily in both normal and pneumonic serum.

An interesting point, and one which may be of theoretical significance, was first noted by Rosenau,[23] and confirmed later by Longcope.[24] This is the production of acid with the formation of a copious precipitation, when pneumococci are grown in pneumonic serum. This acid production does not take place in normal serum. Now, it is well known that the production of acid, when pneumococci are grown in fermentable sugar media, exerts a deleterious influence upon the viability, and probably also upon the virulence of the pneumococci. It is not improbable, though experimental evidence is as yet lacking, that this acid production from some unknown fermentable sugar-containing substance in the serum, occurs regularly during the course of a lobar pneumonia, and is an important factor in checking the growth of the pneumococci, and in attenuating their virulence. That such an attenuation does actually occur is suggested by the fact that virulent pneumococci are insusceptible to phagocytosis outside the body, as shown by Hektoen.[25] There is, as we shall see, a marked and sudden increase in the opsonic substances in the serum towards the end of the disease, and this would be quite without significance did we not suppose that the pneumococci, having lost their original virulence, are thus rendered susceptible to phagocytosis.

This acid production by the pneumococci growing in pneumonic serum is of interest also from another point of view. Opie,[26] working with sterile inflammatory exudate produced by the injection of aleuronat into the pleural cavity of dogs, has shown that suspensions of washed leucocytes, when incubated for several days under toluol, undergo marked proteolysis, the proteolytic ferments being contained within the leucocytes. When, however, serum either from the exudate itself or from the blood, is added to the suspension of leucocytes, autolysis is distinctly inhibited. This inhibitory influence of the serum Opie ascribes to certain substances which he calls anti-autolysins. These anti-bodies upon investigation, proved to be efficient in checking self-digestion of the leucocytes only when acting in an alkaline or neutral medium. When the reaction of the serum was brought to an acidity of 0.2 per cent. by the addition of acetic acid, the autolysis of the leucocytes was no longer restrained.

The application of these facts toward an explanation for the process of resolution in pneumonia, is very tempting. We have

but to suppose that the pneumococci, growing in the lung, acidify the serum in sufficient degree to annul its normal inhibitory influence in checking the autolysis of the leucocytes. As soon as this degree of acidity is reached, the intracellular ferments within the leucocytes exert their proteolytic powers, and, as we say, the "exudate resolves" or digests itself. I may say in this connection that autolysis of washed leucocytes in vitro takes place with equal readiness in a slightly acid or alkaline media. This explanation of the phenomenon of resolution is, of course, entirely speculative. I suggest it merely to point out what an interesting field for experimental research is opened up by Rosenau's observation.

The behavior of the opsonins during lobar pneumonia has been recently studied by Herman Wolf.[27] In a typical case ending in recovery, the opsonic index remains below normal, until within a few hours before the crisis; it then rises rapidly to reaching its maximum within twenty-four hours after the crisis, and again falls gradually towards the normal. In the fatal cases, especially those associated with a hypo-leucocytosis, the opsonic index remains persistently below the normal.

Wolf points out that during the negative phase, although the opsonic power of the individual leucocyte may be reduced, the great numerical increase in the leucocytes more than counterbalances this deficiency.

For example, if in normal serum each leucocyte takes up 4 pneumococci, and there are 6,000 leucocytes to the cubic millimeter; then the leucocytes in 1 cubic mm. of blood would take up 24,000 pneumococci.

In pneumonia during the negative phase, when the opsonic power is below par, and, let us say, each leucocyte can take up but 2 pneumococci, then, nevertheless with a leucocytosis of 30,000, 60,000 pneumococci would be taken up by the leucocytes in each cubic mm. of blood, or almost twice as many as in normal blood. The opsonic curve representing the total opsonic power of the blood, corrected in this way, would show a rise throughout the entire course of the disease.

Such a computation, however, is largely schematic. Even if it were based on experimentation with the homologous organism, the conditions in vitro, especially as regards virulence, must be very different from those that obtain in the living body. Another possibility of error which the author himself has recognized, lies

in the fact that the opsonius may not be developed in sufficient quantity to sensitize all the bacteria until late in the disease.

The treatment of lobar pneumonia with vaccines, after the technique of Wright, has, so far as I know, not been tried on an extended series of cases. Wolf, in the article to which I have referred, reports the result of the injection of heated virulent cultures of pneumococci in 14 cases of lobar pneumonia. Of these, 3 died; 5 defervesced on the third day, 3 on the fourth, 1 on the fifth and 2 on the sixth. Of the 3 fatal cases 2 were senile pneumonias, and one was a severe infection with marked hypoleucocytosis.

These results, so far as they go, seem promising—especially the early crisis on the third day—in the majority of the cases. They certainly justify further experimentation in this line.

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A STUDY OF PNEUMOCOCCI AND ALLIED ORGANISMS IN HUMAN MOUTHS AND LUNGS AFTER DEATH.

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Our studies have been confined especially to a determination of the prevalence of the pneumococcus in normal lungs, and in lungs which presented various lesions. At the same time, we have tried to determine, by experimental methods, how justly one may draw inferences with regard to the flora of the living lung from cultural findings after death.

Numerous investigations have shown that the pneumococcus is more or less constantly present in a variety of pulmonary lesions, especially in lobar pneumonia. The pneumococcus has been isolated from a considerable proportion of apparently healthy lungs, both human and animal, that have been examined after death. A similar micro-organism, or one closely related to it, has been found in the mouths of many healthy human beings

The question of the presence of bacteria in normal lungs has long been an urgent one, because of its obvious bearing upon the determination of the occurrence of the infectious diseases of this organ. If organisms are present in all lungs, it may be supposed that the pneumococcus will be more frequently found in the lungs of patients exposed for some time to a hospital atmosphere. This point, too, we have tried to decide, and for this reason we have classified our cases into the following three groups, viz.:

I. Patients dying outside of hospital.

II. Patients dying in hospital within twenty-four hours of admission.

III. Patients dying in hospital twenty-four hours or longer after admission.

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This report embodies the results of the bacterial examination of a series of forty-two (42) human lungs. The cases were taken at random, so as to comprise a variety of lesions. There were included cases which died in the wards of Bellevue Hospital, and a lesser number of coroner's cases, which died either outside the hospital or in the hospital within twenty-four hours after admission.

These lungs were selected from subjects examined within twenty-four hours after death. The bodies were removed from the wards, usually within an hour after death, and kept in cold storage until examination.

Methods Employed in the Study of the Bacteria in the Mouth and Lungs.

Isolation—The isolation of the organisms studied was carried out in the following manner: The lung to be cultivated was clamped before removal, by a large hysterectomy clamp, which firmly occluded the large vessels and bronchus. This was done to prevent, as far as possible, the aspiration of fluid or mucus from the trachea, during the manipulation of removing the lungs. The surface of the lung was thoroughly seared with a large knife over the posterior portion of the lower lobe. The juice was obtained in the usual manner, by stabbing the sterile area with a heated Nuttall spear, and was then thinly streaked on glycerine-agar plates. The fluid suspension of material from the lungs for the inoculation of the mice was obtained as follows: With sterile forceps and scissors, a large piece of lung was excised from the seared area and cut into small pieces in a sterile Petri dish; in cases where the juice expressed was insufficient a little broth was added. From 0.5 to 1.0 c.c. was inoculated into the skin of the back. In the majority of cases white mice were used, but, owing to the difficulty of obtaining a sufficient number, we were compelled (in a few instances which are noted) to use colored or spotted mice.

From the lung plates, after from twenty-four to forty-eight hours, sub-cultures were made on Loeffler's blood-serum from pneumococcus- or streptococcus-like colonies; usually as many as half a dozen were transplanted.

From the heart's blood of mice dying after inoculation, streak plates were made on glycerine-agar, and the colonies which had

developed after from twenty-four to forty-eight hours were sub-cultured on Loeffler's blood-serum. In case the colonies varied in appearance, several sub-cultures were always made from each type.

For the isolation of the pneumococcus from saliva we have depended almost wholly upon the subcutaneous inoculation of mice with a small amount of mucus from the mouth. This was obtained at autopsy, on a cotton swab, which was then shaken in broth and the suspension injected. The subsequent proceedings were identical with those above described for the lungs. Glycerine-agar plates were made in a few instances, but, on account of the difficulty encountered in obtaining satisfactory plates, this was not done as a routine.

Morphology—The morphology of the bacterial flora of the lung was studied in cover-slip preparations and in sections of lung tissue. As a routine stain for the demonstration of capsules, the two staining methods as described by Hiss* were employed; in smears from the lung tissue, a Gram preparation was also made. Sections were stained for bacteria by the Gram-Weigert method.

Cover slip preparations from the heart's blood of mice, from typical colonies on plates, from the primary sub-cultures on Loeffler's medium, were examined as a routine, and in many instances from the transplantations on different media.

Determination of Cultural Characters—For the study of cultural characteristics, transplants were made from the primary Loeffler sub-cultures upon the following media: Broth, gelatine, glycerine-agar slants, litmus-milk, Hiss' 1 per cent. inulin-serum water (1:3). In addition, the fermentative activities of a certain number of what we may for the present call the different strains of the pneumococcus, as well as the streptococcus, were studied on a series of sugar media containing dextrose, lactose, maltose, saccharose, mannit, or glycogen, and on dextrin and soluble starch.†

Determination of Virulence—This was limited to the inoculation of mice with half or whole cultures, grown for twenty-four

* Journal of Experimental Medicine, 1905, vi, 317.

† The sugars were obtained from Merck. The inulin employed by us in the earlier determinations was obtained from Merck. This inulin was found highly unsatisfactory, from the presence of resistant spores. All our later work was done with Kiliani's inulin. "Eimer and Amend." Many of our earlier cultures were tested again upon this same inulin. Kahlbaum's soluble starch was used for the starch-serum water, in the strength of two per cent.

hours on glycerine-agar slants. Unfortunately, we were not in a position to determine accurately the virulence of the various strains, owing to the insufficient supply of mice at our disposal. Because of this scarcity we rarely were able to ascertain the virulence of the organism in fresh isolations.

Description and Classification of the Pneumococci, Streptococci, and Allied Forms, Found in the Lungs and Mouths.

In consonance with the opinions of more recent observers, we believe that the older criteria relied upon for the differentiation of pneumococci and streptococci are insufficient. Even the fermentative activities upon which so much stress has recently been laid have not been, in our hands, an infallible guide, especially with reference to the identification and classification of the intermediate types of these two closely related cocci, of which many have been encountered.

We must likewise take exception to the older and perhaps still widely prevalent view, that streptococci, unlike the pneumococci, may not produce a bacteriæmia in mice, as in many cases we were able to isolate streptococci in cultures from the heart's blood of mice taken immediately after death.

In order to describe the bacteria we have encountered during the course of this work we have been forced, somewhat against our will, to adopt an arbitrary classification founded mainly upon the variations in morphological and physiological characters. The most important and constant of these are the presence or absence of capsules, and the fermentation or non-fermentation of inulin. Our grouping of these cocci is as follows:

Group I.—*Typical Pneumococci*—By this, we designate those diplococci which ferment inulin with acid formation, and coagulation of the serum; which possess readily stained capsules, not only in the blood of mice, but on various media; which give a diffuse cloudiness in broth, and grow as fine, colorless, delicate, translucent colonies on glycerine agar, and which are virulent to mice. We have met with a number of organisms which, although fermenting inulin with slight acid production, do not coagulate the serum. This, we consider, indicates, in general, feeble growth.

Group II.—*Streptococcus Mucosus*—By this term, we refer to a series of organisms which differ from the typical pneumococcus in their abundant, moist, or mucous growth upon various solid media and in the more constant production of chains upon media, but which resemble the pneumococcus in possessing a constantly demonstrable capsule, in their fermentative activity, and in their virulence to mice.

In conformity with the observations of other observers, we have found that the capsules of these diplococci are more easily demonstrable than those of the typical pneumococcus, and that there are certain variations in the structure of the capsules, which we have not observed in those of the pneumococcus. We have found that the cocci of this group grow more constantly and abundantly at room temperature in gelatin.

We have not as yet been able to decide definitely, whether the group constitutes a distinct variety of the pneumococcus, or whether the mucous character of the growth and chain formation is dependent upon variations in the media. Thus we have found that on dry Loeffler's serum tubes the growth of these organisms may be dry, whereas on wet tubes it tends to be mucous-like in character. We have also seen organisms resembling the pneumococcus, after passage through mice, assume a viscid character of growth. In fact, there seem to be numerous gradations between these two types in this respect.

Group III.—*Diplococci Resembling Pneumococci which Possess no Demonstrable Capsule*—No capsules have been demonstrated in these diplococci, either in the heart blood of mice, or on culture media after repeated examination. They produce acid in inulin, but do not coagulate the serum. In all other respects, they resemble pneumococci.

Group IV.—*Diplococci Resembling Streptococci which Possess no Demonstrable Capsule*—No capsules have been demonstrated in these diplococci, either in the heart blood of mice, or on culture media. They are active inulin fermenters. These cocci, although fermenting inulin with acid production, have greater points of similarity to the streptococcus than to the pneumococcus. They grow abundantly in artificial media, produce clouding, or flocculi and granules in broth, grow readily at room temperature, and show a marked tendency to chain formation.

Group V.—*Typical Streptococci*—These have no capsules, do not ferment inulin with acid production, and grow readily at room

temperature. They produce clouding in broth, or form granules or flocculi adherent to the sides of the tubes.

Sources of Error in Technique.

It is obvious that there are many possibilities of error in the isolation of pneumococcus-like organisms from the lungs by methods above detailed. On account of the similarity in the colonies on glycerine-agar, and even on ascitic-agar, there is always difficulty in distinguishing on inspection pneumococcus from short-chained streptococcus colonies, and in consequence the pneumococcus, though present in small numbers, may escape detection, for it is obviously not practical to sub-culture more than a limited number of suspicious colonies.

Overgrown plates are another but not a frequent source of trouble. The organisms responsible for this are not only those which are introduced through natural errors in technique, but also those which we have found more or less constantly present in the lungs cultivated after death, as verified by a thorough examination of cover-slip preparations from the lung tissue.

Again, another source of error lies in the natural insusceptibility of white mice, and even more so of certain strains of colored mice, to infection by pneumococci. Thus it has been found by us that colored mice are more resistant to infection than white mice when inoculated with similar doses. Morever, it has been found that in some of our cases even a white mouse does not succumb to the inoculation of a comparatively large amount of expressed lung juice, although cover-slip preparations of the lungs have shown typical capsulated diplococci, positive to Gram. For this reason, so far as it has been possible, we have inoculated two mice from each case.

Still another though less frequent and serious difficulty is that encountered in the isolation of pneumococci in streak agar plates from the heart blood of mice dying of mixed infection. In a certain number of cases we have found Bacillus mucosus capsulatus to be the predominating organism; in other cases, non-capsulated cocci which we have not as yet identified. In a certain number of cases also, though pneumococcus-like organisms were present in smears from the lung, the mice inoculated died from streptococcus, Bacillus mucosus capsulatus, or other infections. On the other hand, we have encountered capsulated cocci in the heart's blood of mice, as well as upon our lung plates,

which failed to grow on transplantation upon glycerine-agar or Loeffler's blood-serum.

With this brief introduction, we can now present more readily the result of our work largely in tabulated form. The tables which follow must be briefly described.

The first column of the tables, headed "Case Number," indicates the number of the case in our series. The next column, the "Accession Number," refers to the entry number of each case in the autopsy accession book of the hospital. The Roman numerals I., II., III., IV., and V. are those we have used to designate the five groups into which we have divided the pneumococci, streptococci, and the cocci allied to them.* The addition sign (+) or the zero sign (o) opposite each case indicates that a coccus of this group has or has not been isolated.

* I.—Pneumococcus.
II.—Streptococcus mocosus.
III.—Diplococci without capsules, which ferment inulin only with acid production.
IV.—Diplococci without capsules, which are active inulin fermenters, and which closely resemble streptococci.
V.—Streptococci.

Pneumococci, Streptococci, and Allied Micro-organisms in Mouths after Death.

In fourteen cases the mouths were cultivated by the methods sufficiently described above. We append the analysis of our findings:

Table I.

Analysis of Pneumococci, Streptococci, and Allied Micro-organisms, Isolated from the Mouth After Death.

Case No.	Accession No.	Group I.	Group II.	Group III.	Group IV.	Group V.	Unidentified.
1	82	+	o	o	o	o	(?)
2	98	o	o	o	o	+	o
3	105	o	o	o	o	+	o
4	110	+	o	o	o	o	o
5	153	o	o	o	+	o	o
6	164	o	o	o	o	+	o
7	165	+	o	o	o	o	o
8	166	+	o	o	o	o	o
9	167	o	o	o	o	o	+
10	170	o	o	o	o	+	o
11	172	o	o	o	o	o	+
12	191	+	o	o	o	+	o
13	199	o	o	+	o	o	o
14	233	o	o	o	o	o	o
Total, 14 cases....		5	0	1	1	5	2

The pneumococcus, as shown by Table I., was isolated five (5) times, the streptococcus five (5) times, and intermediate organisms twice. Of these twelve cases, all but three were in the hospital over twenty-four hours. Two died with lobar pneumonia; in one of these, the mouse injected with mouth secretion died of streptococcus infection, and this organism, in pure culture, produced a bacteriæmia fatal within twenty-four hours in a mouse inoculated with three-fourths of a glycerine-agar growth twenty-four hours old.

The virulence of the cocci isolated was tested in eight (8) cases. Three (3) of these were pneumococci, four (4) were streptococci, and one (1) Group IV. One of the streptococcus

cultures proved to be non-virulent. All the others killed the mice in from eighteen to forty-eight hours, partial or whole cultures being inoculated.

All the mice inoculated with mouth secretion died, in most cases, within twenty-four hours. Mice inoculated with the secretion from two cases died with infections other than pneumococcus or streptococcus. From a third case, one of the mice inoculated died after four (4) days, no organisms being recovered in smears or by culture; the second mouse of the same case remained alive.

These few observations seem to show that the streptococci isolated from the mouth were quite as virulent as the pneumococci. The pneumocci obtained from two (2) cases not dying of lobar pneumonia were found to be quite as virulent as those obtained from pneumonic lungs post-mortem. Further conclusions, however, as to the general virulence of the pneumococci obtained from the mouth do not seem to be justified.

We have made no attempts to determine the frequency of pneumococci in the mouths of ward patients or attendants on account of an insufficient supply of mice.

Pneumococci, Streptococci, and Allied Bacteria Isolated from the Lungs after Death.

Forty-two (42) cases were examined. The analysis of our findings follows in tabulated form (Table II.).

Table III. shows the number of cases in which the various groups of cocci have been found, either alone or in association with the other groups.

We are now in a position to analyze the facts presented above, in Table IV., which requires a brief explanation. The cases cultivated in this research have been divided into three classes, according to whether the lungs were normal in the gross or presented the lesions of lobar pneumonia, or were the seat of a variety of other lesions. Each of these classes has again been divided into three classes, viz., those cases dying outside the hospital, those dying in the hospital within twenty-four hours of admission, and those dying twenty-four hours or more after admission.

TABLE II.

Analysis of the Diplococci Isolated from the Lungs

Case No.	Accession No.	Group I.	Group II.	Group III.	Group IV.	Group V.	Unidentified.
1	43	o	o	o	o	o	
2	45	o	o	o	o	–	
3	46	o	o	o	o		
4	49	+ (?)	o	o	–		
5	52	o	o	o	o	o	
6	54	o	o	+		–	
7	63	–	o	o		o	
8	68	+ (?)	o	o		–	
9	70	+ (?)	o	o		o	
10	71	o		o		o	
11	82	+	o	+		+	
12	98	o	o	o	o	–	
13	102	o	o	o	o		
14	105	+ (?)	o	o	–		
15	110	+	o	o		o	
16	113	o	o	o	o	–	
17	114	+ (?)	o	o	o		
18	123	o	o	o	–	o	
19	140	+	+	+	o	o	
20	147	o	o	o	o	o	
21	149	o	o	o		o	
22	153	o	o	+	o	o	
23	160	o	–	o	o	o	
24	164	o	o	o	o	–	
25	165	–	o	o	o	+	
26	166	+	o	o	o	+	
27	167	o	o	o	o	+	
28	170	+	o	o	o	+	
29	172	+	o	o	o	o	
30	175	o	o	o	o	+	
31	191	+	o	o	o	o	
32	199	o	o	+	o	+	

* See explanation of Table I., p. 113.

† The cocci of Group I. which are marked (?) were not absolutely identified as pneumococci, but they in all probability belong to the group, and they have therefore been included.

Case No.	Accession No.	Group I.	Group II.	Group III.	Group IV.	Group V.	Unidentified.
33	C I	+	o	o	+	o	
34	C II	o	o	o	+	+	
35	C III	+	o	o	o	o	
36	C IV	+	o	o	o	o	
37	C V	o	o	o	o	+	
38	C VI	o	o	o	o	+	
39	C VII	+	o	o	o	o	
40	233	+	o	+	o	+	
41	237	+	o	o	o	o	
42	258	+	o	o	o	o	

TABLE III.

Analysis of the Diplococci Isolated from Lungs.

Group of Organisms.*	No. of Times Found.	Accession N. of Cases.
I alone.	10	68, 70, 110, 172, 191, C III, C IV, C VII, 237, 258.
II "	2	71, 160.
III "	1	153.
IV "	3	52, 54, 149.
V "	10	45, 46, 98, 102, 113, 164, 167, 175, C V, C VI.
I + II	o	
I + III	1	82.
I + IV	1	C I.
I + V	5	105, 114, 165, 166, 170.
II + III	o	
II + IV	o	
III + V	o	
III + V	1	199.
IV + V	1	C II.
I + III + IV	1	63.
I + II + III	1	140.
I + IV + V	1	49.
I + III + V	1	233.
Unidentified	2	123, 147.

* See Explanation of Table I.

The data presented in Table IV. may be thus briefly reviewed:

I.—NORMAL LUNGS.

a. Two Cases Dying Outside of Hospital—In one, the pneumococcus was isolated; in the other, only a streptococcus.

b. Four Cases Dying within Twenty-four Hours of Admission—In these (63, 68, 153, 223) the pneumococcus was isolated three times; one of these cultures (68), however, may be considered questionable.

It is interesting to note that likewise in three of the cases organisms of Group III. (non-capsulated inulin fermenter) were found, and in two of the cases streptococci (Group V.).

c. Eight Cases Dying Twenty-four Hours or More after Admission—The pneumococcus was obtained in two cases: in 166, where it was associated with streptococcus (Group V.); and in 258, where it occurred alone. In two cases (71 and 160), Streptococcus mucosus (Group II.); in one case (149), Group IV.; in four cases (98, 166, 167, C V.) streptococci (Group V.).

The pneumococcus, if 68 be called a true pneumococcus, was thus isolated five times. It is, perhaps, noteworthy, that pneumococcus was isolated from the cases dying outside, and those less than twenty-four hours in hospital, in four out of six cases, 66%, whereas it was isolated in only two out of eight cases which had been in the hospital over twenty-four hours, 25%.

TABLE IV.

Analysis of the Cases, According to the Groups of Diplococci and the Lesions Found in the Lungs at Autopsy and also According to the Length of Time of the Cases in the Hospital.

	Normal.						Lobar Pneumonia.						Other Pulmonary Lesions.						Total.					
	No. of Cases.	Group I.	Group II.	Group III.	Group IV.	Group V.	No. of Cases.	Group I.	Group II.	Group III.	Group IV.	Group V.	No. of Cases.	Group I.	Group II.	Group III.	Group IV.	Group V.	No. of Cases.	Group I.	Group II.	Group III.	Group IV.	Group V.
Not in Hospital	2	1	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	3	2	0	0	0	1
In Hospital 24 hours or less	4	3	0	3	1	1	3	1	1	2	0	1	2	2	0	0	1	0	9	6	1	5	2	2
In Hospital over 24 hours	8	2	2	0	1	4	9	5	0	0	0	6	13	6	0	1	4	6	30	13	2	1	5	16
Total	14	6	2	3	2	6	13	7	1	2	0	7	15	8	0	1	5	6	42	21	3	6	7	19

Summary of Table II

Total number of cases cultured	42
Micro-organisms obtained in	41
Group I.	21 (5 doubtful).
" II.	3
" III.	6
" IV.	7
" V.	19
Unidentified	2

II.—LOBAR PNEUMONIA.

Thirteen (13) cases, one dying outside the hospital, three (3) within twenty-four hours of admission, and nine (9) after residence in hospital of twenty-four hours or over. The Pneumococcus was isolated from seven of these cases; this includes one non-inulin fermenter (114), which, however, exhibited a capsule after the second passage through mouse, the culture having been taken from the pneumonic lobe.

Cultures were made in seven of the thirteen cases from the consolidated lobe; in two, from the non-consolidated. Of the remaining four (4) cases, which showed organization of the exudate (so-called organizing pneumonia), cultures were made from the involved lobe three (3) times; from the uninvolved lobe, once.

From the seven lobes showing a typical pneumonia with exudate, in three cases (140, C VII., 237) typical pneumococcus (Group I.) was obtained; once (140) associated with Streptococcus mucosus (Group II.); in two cases (105, the organism growing poorly, and 114, above mentioned) two typical pneumococci were isolated, associated with streptococci (Group V.); in another case (199) Group III., and also a streptococcus, Group V.; in another (113) Group V. was isolated, no other organisms being found in this case.

Of two cases cultivated from the uninvolved lobes, one (110) gave a typical pneumococcus; the other (147) showed B. muscosus capsulatus in pure culture from the lungs in plates, and from the mouse in plates, no pneumococci having been found in the cover-slip preparations made from the heart's blood of mouse.

In three cases where the lobes with organizing pneumonia were cultured, in one (43) no bacteria were found in plates, the single mouse inoculated remaining alive; in two cases (46 and 102) streptococcus (Group V.) alone was isolated.

In one case of organizing pneumonia (165), in which the involved lobe was cultured, a pneumococcus (Group I.) and a streptococcus (Group V.) were isolated.

III.—LESIONS OTHER THAN LOBAR PNEUMONIA.

Of the fifteen remaining cases, none died outside the hospital; two were in hospital less than twenty-four hours; in one of these (C I.) pneumococcus and an organism of Group IV. were found; in the other (C III.) pneumococcus alone was found.

Of the thirteen cases over twenty-four hours in hospital, the pneumococcus was found in four cases; in two cases (49 and 70) organisms, probably pneumococci (Group I.), were obtained. In Case 49, organisms of Groups IV. and V. were also isolated; in 70 no other organisms were found. In one case (123) an unidentified, non-capsulated diplococcus, which did not grow on transplantation from the lung plates, was obtained; the mouse inoculated with lung tissue remained alive.

From the tabulated summary of Table IV we see that the total number of cases cultured was forty-two, and that in all but two (2) cases micro-organisms were obtained. From these cases, pneumococci and streptococci were obtained in practically similar percentages—that is, in 50 per cent. These results, as far as the pneumococcus is concerned, agree with those obtained by Beco, 50 per cent. pneumococci and 45 per cent. streptococci in human lungs.* Boni,† in lungs of pigs, has also obtained similar percentages. Dürck,‡ in pneumonic lungs of children, obtained pneumococcus in cases of lobar pneumonia, some of which were organizing or unresolved pneumonias, in seven out of thirteen, 52 per cent., streptococci being found in the same proportion.

Discussion of the Significance of the Data above Summarized—A consideration of these facts suggests a number of interesting questions: Are the pneumococci present in the exudate of lobar pneumonia similar to the pneumococci which have been found in the mouths of normal persons, presumably as harmless inhabitants? If so, they reach the lungs either through the lymphatics or the vascular channels at some time during life, or by inhalation, lodging there as harmless saprophytes until some as yet unknown change in their host causes them to acquire increased virulence. A second possibility is that pneumococci of greater virulence than those found in the mouth are inhaled with the air current during life, determining the onset of the disease. However, it is still an open question whether pneumococci are present in the lungs of normal persons during life. May not their presence in normal lungs, as found by post-mortem examinations, be explained by a terminal septicæmia, such as has been established for the streptococcus and other micro-organisms, or do the pneumococci of the mouth gain access to the air vesicles with the saliva, either aspirated during the death agony or, later, by gravi-

* Beco, Archiv. d. la medicine expérimentale, 1889, xi., 317.
† Boni, Deutsch. Arch. f. klin. Med., 1901, Bd. 69.
‡ Dürck, Deutsch. Arch. f. klin. Med., 1897, Bd. 58.

tation consequent upon the manipulation of the body after death, in its transportation from the wards to the morgue?

These problems complicate the interpretation of the various factors concerned in the production of lobar pneumonia. With the more theoretical aspects of the question, such as the disturbance in balance between the host and the inciting agent of the disease, we have not attempted to cope, except in so far as we have roughly determined the virulence for mice of the organisms isolated in our mixed series of cases.

The statistics obtained by the investigators mentioned above and by ourselves might lead one to the inference that bacteria of the pneumococcus or streptococcus group exist during life in practically all lungs, whether normal or diseased. But, as we have already suggested, grave theoretical objections may be urged against such a conclusion. Though we have been able to obtain these organisms in 97% + of our cases, the possibility, as suggested above, that the pneumococci so frequently present in the mouth may reach the lungs by aspiration during the death agony, or after death, during the transportation of the bodies from the wards to the morgue, is no fanciful one.*

The mouth and nose are frequently found at autopsy filled with fluid admixed with frothy and bloody fluid, or vomitus. It is quite evident, under such conditions, that, by mere force of gravity and the fluid communication existing between the upper and lower air-passages, micro-organisms may find their way from the mouth to the small air spaces; or, again, by the compression of the chest wall in moving the body a false respiratory excursion may take place, causing the replacement of the air in the

* Because of its possible bearing upon the gravitation of fluid from the buccal cavity to the lungs, it seems worth while to describe in detail the various manipulations to which the body is subjected during the interval between death and post-mortem examination. After being formally pronounced dead by a member of the house staff, the body is rolled in a shroud, the jaw supported by a four-tailed bandage, and cotton plugged into the mouth and other orifices. Within an hour or less, the cadaver is ready for transportation to the morgue. It is lifted by the shoulders and heels, to a four-wheeled truck, which has been rolled to the bedside. During this manœuvre, with the sagging of the body the head is of necessity at a higher level than the thorax. From the ward, the body is wheeled directly to the morgue,—a distance of about 150 yards. Then again it is lifted from the truck to a wooden frame, which is placed upon the floor next to the carriage, and lying upon this, it is raised, feet foremost, to a compartment in the cold-storage chamber. Here it remains until removed to the autopsy room. In this final handling of the body, the wooden tray is slid from the compartment, the head lowest, and the body is carried upon this wooden frame to the adjacent room. The body is then rolled upon its side, abruptly jolted, and the head raised and lowered several times. The entire proceeding, therefore, involves considerable disturbance of the body before the necessary examinations are made.

lungs by fluid from the mouth and trachea. If this takes place, it then seriously vitiates the value of inferences drawn from cultural findings after death, when applied to the living lung.

In order to determine the frequency of this occurrence by experimental methods, we have had recourse to rabbits, which have been given small doses of broth cultures of B. prodigiosus just before or after death. Furthermore, we have introduced broth cultures of B. prodigiosus (a half drachm or less) into the mouths of patients who had died in the wards, but before removal of the bodies to the morgue.

The method which we have used for the isolation of B. prodigiosus in the mouths and lungs of rabbits and cadavers was the following: The mouth was scraped with a sterile cotton swab; a suspension was then made in melted agar tubes, and plates with one dilution poured. The mouth plates were made in every case in order to make sure that the patients had received the culture, and thus they served as control for the lung plates. From the lung a considerable amount of expressed juice, obtained by squeezing and crushing a piece of excised lung, was distributed in melted agar tubes and pour-plates made. Both series of plates were then kept at room temperature and observed until the appearance of typical red colonies, or for several weeks. Abundant growths of various organisms were regularly obtained in these plates. In no case have we seen that the development of numerous colonies has inhibited the pigment production.*

In nineteen (19) cases, half-drachm or smaller portions of broth cultures of B. prodigiosus were introduced into the mouth by the interne after having pronounced the patients dead. Characteristic red colonies of B. prodigiosus developed on the pour-plates from the mouths of these nineteen cases, and in ten cases more or less numerous colonies of B. prodigiosus developed in the lung plates. In other words, in a little over 50 per cent. of the cases evidence was obtained that micro-organisms introduced into the mouths of patients after death enter the lungs. In half of the cases, as seen from Table V., numerous colonies developed on the lung plates.

* A few control experiments were made, to test this point; tubes were inoculated with varying proportions of dysenteric fæces, and broth cultures of B. prodigiosus. These showed that pigment formation by this culture of B. prodigiosus was not inhibited by the excessive growth of other bacteria.

Since it was impossible to personally control the giving of the cultures after death, we have not included in the table nine cases in which the mouth plates were negative. We, however, believe that of these nine cases some received the cultures, but that the B. prodigiosus failed to develop its characteristic color upon the mouth plates. We have positive knowledge that this occurred in one case (279, not included in table), the cotton swab of the mouth being stained pink. Moreover, as above stated, our B. prodigiosus developed pigment in overcrowded plates; nevertheless, as is well known, pigment formation is subject to a number of disturbing factors which are not readily controlled or perhaps even known.

In support of this view it may be stated that in one case, 175, abundant colonies developed in the lung plates, whereas none appeared in the mouth plates; this case has not been included in the final estimate of the percentages of positive results. For these reasons, we believe that the percentages given are too low for the mouth as well as for the lung plates.

It is certainly a striking observation that likewise in about fifty per cent. (50%) of our forty-one cases the pneumococcus was isolated. It seems a fair inference from this coincidence that the utmost caution must be observed in the interpretation of the cultural findings of the lung from post-mortem statistics.*

* In five (5) cases, the prodigiosus culture was received shortly before the exitus lethalis. In one case (82, alcoholic delirium), numerous colonies developed in both the mouth and the lung plates. In two cases (98, 105), the mouth plates alone developed colonies, the lung plates being negative. In case 110, negative results were obtained in the mouth and lung plates; this, however, is readily accounted for by the fact that the culture was administered twenty-four and, again, fourteen hours before exitus. Another of the negative cases (C V), where no colonies developed on the lung plates, is readily enough accounted for by the closure of the glottis from œdema, after a cut-throat wound of the neck. Thus, colonies of prodigiosus were obtained from the lungs in one out of three cases, 33 %, if the above reasons are valid for excluding the two cases (110 and C V). As regards these experiments, however, only negative results are of value, since one cannot make sure that the organisms introduced during life did not reach the lung after death.

We append below the statistics in tabulated form:

TABLE V.

Table Showing the Cases in Which B. Prodigiosus, Introduced into the Mouth Post-mortem, was Recovered from the Mouths and Lungs.

No.	Mouth.	Lung.	No.	Mouth.	Lung.
166	—	—	214	+++	+
168	+	+++	224	+	—
171	+++	—	233	+++	+
172	+	+	241	+++	—
181	+++	+	258	+++	—
188	+++	++	262	+++	+
199	+	—	278	+++	—
201	+++	+++	281	++	+
205	+	—	288	+++	+++
206	+	—			

Total number of cases	19
Mouth positive	19
Lung positive	7

Our work upon the entrance of B. prodigiosus from the mouths of rabbits to their lungs has been unfortunately confined to two (2) rabbits. One rabbit was given 0.5 c.c. of a broth culture of B. prodigiosus when under ether. The rabbit died twenty minutes later without gasping. The body was then slanted upon a tray with elevated head. (Post-mortem, twenty-nine hours later.) No colonies of B. prodigiosus developed in either mouth or lung plates.

The other rabbit, dying from an intraperitoneal inoculation with the meningococcus, was given 0.5 c.c. of a broth culture, and died slowly several hours later. At autopsy, performed at once after death, pour-plates were made from the mouth and lungs. Numerous colonies developed on the mouth plates; the lung-plates failed to show red colonies. We abandoned this line of experimentation for the reason that the conditions so obviously differ from those of the human respiratory tract, which almost constantly contains more or less fluid, at least after death.

The Agglutinating Reactions of the Pneumococcus, Streptococcus, and Allied Groups of Diplococci.

The impracticability of using agglutinating reactions, especially difficult in this group of cocci, led us to forego this aid to species identification. This method, as has been repeatedly shown, when used upon species belonging to the same group, has no differential value, unless very tedious and exact quantitative serum tests are employed. For these reasons, and because of the impossibility of obtaining sera of high valency within the limited time at our disposal, we could not avail ourselves of this method during the greater part of this investigation.

Before giving our conclusions, we deem it best to give the results that we obtained upon the agglutinability of cultures of species belonging to the various groups into which we have divided the various diplococci that we encountered during the progress of our work.

We were able, however, at the end of our work, to make use of the method of obtaining mass cultures for agglutination tests described by Prof. Hiss, which, in his hands, has yielded such brilliant results. With a rabbit immune serum giving a complete agglutination at 1:400 with the homologous pneumococcus, which we obtained through the kindness of Prof. Hiss and Dr. Borden, the following tests were made the day after receipt of the serum, which was from a recent bleeding:

Broth cultures were made of species of each of our five (5) groups of cocci, strict attention being paid to the details as given in the method described by Hiss, in the article above referred to. The results of our agglutination tests obtained with the pneumococcus rabbit serum, mentioned above, and with a normal rabbit serum, may thus be briefly described. Two diplococci of Group I. were used; the first 230, a typical pneumococcus, was obtained from a case of suppurative sphenoiditis at autopsy. This pneumococcus did not lend itself to the agglutination test, at least with the mass culture we employed since, the control settled out promptly, and clumps were speedily developed. The second diplococcus, C VII., another typical pneumococcus, was not clumped by the normal rabbit serum, after twenty-four hours, the control showing only a few small, microscopic clumps. With the pneumococcus serum, large flocculi developed in the 1:10 dilution, but the reaction did not become complete, even after twenty-four hours, the fluid still being cloudy. Small flocculi were, however, found in the 1:200, as well as in the 1:600 dilution, unlike the control.

Only one diplococcus of Group II. was tried, namely 71, a typical streptococcus mucosus, which gave a slight reaction only in the lowest

* Journal of Experimental Medicine, 1905, vii., 223.

dilution, namely, 1:10. Normal rabbit serum gave no reaction in a similar dilution.

One diplococcus of Group III. was tried (199). It gave, with the pneumococcus rabbit serum, a positive reaction at 1:100, but no complete reaction was obtained even at 1:10, the fluid becoming only slightly clearer with small clumps on the sides of the tubes. The reaction in the 1:10 and the 1:100 dilutions differed only in the size of the clumps. Normal rabbit serum had no action upon this diplococcus.

Three organisms of Group IV. were tried. Culture 49a gave a slight reaction with pneumococcus serum, in the 1:10 dilution, the clumps being larger than those of the control. After twenty-four hours, all the dilutions and the control, however, became practically clear. Culture 149 was not affected by the pneumococcus or the normal rabbit serum in any dilution. With culture C H. the dilutions and the control were practically alike, although possibly there were larger granules in the lowest dilution than in the control.

Two organisms of Group V., typical streptococci, were tested. Culture 214 S did not lend itself to the agglutination test, on account of the prompt development of large granules, and the rapid clearing of the fluid in the tubes in the control, as well as in dilutions. With the other streptococcus, 153, larger granules were found, and a quicker settling in the lower dilutions occurred than in the control, which, however, after twenty-four hours, became perfectly clear.

Thus, if one may judge from a single examination, the method of mass cultures or agglutination tests have shown that only in the case of the pneumococcus, C VII., positive agglutinations were obtained, and even with this organism no complete reaction was given in the lowest dilution, namely, 1:10, after twenty-four hours, with the possible exception of the diplococcus 199, of Group III.

It seems to us somewhat forced to draw any conclusions, from the single agglutination test described above, upon the method of using mass cultures, but we believe that the test shows that the diplococci which we have not identified as pneumococci—in other words, all those belonging to other groups than Group I.—have no or very slight agglutinating affinities to the pneumococcus.

Before concluding, it may be well to state that we fully recognize the arbitrariness of our method of classifying the diplococci belonging to the pneumococcus and streptococcus groups.

The experience we have gained in the examination of a large number of cultures convinces us that it is not justifiable to consider all diplococci which ferment inulin, pneumococci. Thus, C H. and 149 and 49a are rapid inulin fermenters, but in all other respects they resemble streptococci. It is of interest to note in con-

nection with 49a, that its property of fermenting inulin is a most variable one. Thus, on passage through the first mouse, after its primary isolation from the lungs upon glycerine-agar, it lost its fermenting action upon inulin, only to regain it on further cultivation upon serum water made from the same stock of inulin Further passages through mice did not affect this function. Again, later cultures of 49a lost their capacity to ferment inulin, but regained it on transplatation, and are now active and rapid fermenters. Cultures C II. and 149 have never changed, having always been active fermenters.

We consider that the presence of capsules like the inulin reaction cannot be regarded as an infallible guide for the differentiation of these diplococci, especially when the Hiss capsule methods are employed, for the reason that we have encountered streptococci with distinct capsules.

The cocci of what may be called the intermediate groups, III. and IV., it may be of interest to state, have been found in the blood during life, and have been recovered from the pial exudate of cases of meningitis. A further study of these interesting diplococci is greatly needed.

Conclusions.

The following conclusions may be drawn, based upon the result of our researches:

1. Organisms of the pneumococcus or streptococcus group are present in the lungs of practically all cases, whether normal or showing a variety of lesions; strictly speaking, they were found by us in forty out of forty-two cases, or in 95 per cent. of our series.

2. The pneumococci and the streptococci were obtained in practically similar percentages—that is, in 50 per cent. of the cases.

3. Pneumococci were not obtained more frequently in the small series of patients exposed for some time to hospital atmosphere; our tables show the contrary to obtain. The number of cases examined were, however, insufficient, and the findings may thus be accidental, and hence of no value.

4. Test micro-organisms, namely, small portions—half a drachm or less—of B. prodigiosus, introduced into the human

mouth after death, were conveyed to and recovered from the lungs by culture in a little over half of the cases in which this experiment was tried. The test micro-organisms are, we believe, conveyed to the lungs with the fluid which collects in mouths of persons after death, and which in many cases collects just before death. The numerous manipulations entailed in the removal of the body from the wards to the morgue greatly facilitate the entrance of any fluid from the pharynx and buccal cavity into the lungs.

It follows logically, from the results obtained in this experiment, that the cultural findings after death are no guide to the bacterial contents of the lungs during life, and that any deductions made from such findings are unreliable and deceptive. Granted that our explanation be correct, there is every reason to believe that any of the micro-organisms present in the mouths and pharynx and in many cases in the stomach contents may enter the lungs and, if the conditions be suitable, increase in numbers, during the time between death and the examination of the lungs.

5. There exists, perhaps, more frequently than has hitherto been suspected, a series of diplococci, intermediate between the typical pneumococci and streptococci. The diplococci of this type have been found in forty (40) per cent. of our cases.

The differential diagnosis of these atypical diplococci from the pneumococci and streptococci is a difficult one, depending, as it does, upon general cultural characteristics. No single character, such as the presence of capsules or the fermentation of inulin, virulence, etc., has been found to be a certain criterion. The few agglutinative reactions we have made seem to show that these intermediate diplococci, those of Groups II., III., and IV., have no or only slight agglutinative affinities to the typical pneumococcus. Further tests must, however, be made with the various methods at our disposal before this statement can be accepted as final.

These diplococci are of interest from the fact that they have been found in the blood during life, and in the pial exudate of cases of meningitis, endocarditis, etc.

6. Our studies have thrown no light whatever upon the conditions which determine the onset of lobar pneumonia in apparently healthy persons. Moreover, we have been unable to

draw conclusions as to the presence of pneumococci in the lungs during life, or as to the channels by which they gain access thereto.

In concluding, we gladly avail ourselves of the opportunity of acknowledging our indebtedness to Prof. T. Mitchell Prudden for help and advice freely given in the course of this work, and to the various members of the house staff of Bellevue Hospital for their aid and assistance in the pursuit of these researches, especially to Dr. Frank Erdwurm.

THE PRESENT TREATMENT OF PNEUMONIA AS EXEMPLIFIED BY THE ROUTINE TREATMENT OF THE DISEASE IN FOUR OF THE LARGE NEW YORK HOSPITALS.

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The fact that penumonia to-day occupies first place in mortality statistics as the cause of death arrests our attention. In New York the statistics of the Board of Health show that the mortality curve of pneumonia has passed that of tuberculosis, and more people die from pneumonia every year in Greater New York than from consumption, which until the last few years has always been considered as the great scourge. A satisfactory explanation of the increase of pneumonia is very difficult. Has the type of the disease increased in severity? Have the local conditions favoring its development become worse, or has the treatment of this disease known no advance? These are questions that must interest every medical man. Personally, I believe the treatment of pneumonia at the present day is the least satisfactory of that of any of the acute diseases.

In our large metropolitan hospitals we may look more than anywhere else for the most advanced methods of treatment, as here every new suggestion in therapeutics is tried, weighed, discarded, or adopted. Accurate statistics are kept, thus furnishing data for approximately reliable and impartial conclusions, and all under the critical eye of the young and enthusiastic members of the house staff, who before their two years are up are well able to determine the treatment producing uniformly the best results. Therefore, in the large hospitals I should say we can find, if not the best plan of treatment, certainly the one that is accepted by the majority of the profession as giving the best results. I have taken for comparison four of the largest hospitals

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in New York—the Presbyterian, Roosevelt, New York, and Bellevue.

On the visiting staff of these four institutions are some of the ablest men in the profession, certainly men with large practical experience. As the medical service in all these hospitals rotates, the criticism might be offered that the treatment would change as each new visiting staff comes on duty, in accordance with their personal ideas. This I have found not to be the case to any extent. After a talk with the present house staffs of all these hospitals, I find that there is in each institution what might be called a routine treatment for most of the common diseases, based upon past experience and drawn from the most effective suggestions of the various visiting physicians.

The hospital treatment which I shall speak of may differ somewhat from the treatment we each might carry out, but it will certainly represent the prescribed treatment of the majority. To obtain the details of treatment from the different hospitals, I had typewritten a careful series of questions; these were answered in writing by the different house physicians. Then personal interviews with the medical staff of each hospital were had, and I found that in many instances one or more of the visiting staff had verified the answer to the questions and had added elaborations of their own. I was surprised to find after investigation what a uniformity there was in the treatment of pneumonia in the different hospitals. This is not seen in any other disease, with possibly the exception of acute rheumatism. I will give briefly the general hospital treatment of pneumonia and note the exceptions as I go along.

Cathartics—When a patient enters any one of these four hospitals suffering from pneumonia, he is given calomel, usually in small doses, often repeated, and this is followed by a saline the following morning, generally Epsom salts. The calomel is repeated during the course of the disease if necessary. This as a routine treatment.

Local Applications to Chest—All the hospitals at the present time apply local measures to the chest over the affected lung *only* when the pain and distress is intense. They make no local applications as a routine practice. When applications are made large flaxseed poultices enveloping the chest are applied, although at the New York Hospital and on the first division of Bellevue Hospital ice poultices are sometimes used. The old-fashioned

pneumonia jacket, made by covering flannel with oiled silk, is used in none of the hospitals at the present time for adults suffering from pneumonia. At the New York and Bellevue its use is confined to children.

Diet—All the four medical divisions of Bellevue Hospital put their patients during the height of the disease on an exclusive milk diet, giving the milk plain or modified. The other hospitals make milk the principal article of diet, and alternate it with broths and egg-albumen. Roosevelt Hospital adds eggs and crackers, and there the patients are urged to drink a very large quantity of water.

Fever—The treatment of the fever of pneumonia in the different hospitals varies more than anything else. A temperature of 104° and over is not necessarily an indication for special treatment unless it is accompanied by nervous symptoms, marked restlessness, or delirium; then it is treated as follows in the different hospitals:

Presbyterian Hospital—They use either cold packs to the anterior portion of the chest, the technique of which consists in enveloping the chest with sheets wrung out of water at a temperature of 50° and repeating the application for ten minutes, or by making use of what is known as an "anterior pack." This consists in covering the patient with a sheet and sprinkling with water from a sprinkler, or by a brush wet with water; the temperature of the water is just as it comes from the hydrant, about 85°. This method of reducing the temperature is also used at Roosevelt Hospital.

At the New York Hospital the temperature is controlled by alcohol sponges, and if these are not effective, with a cold pack.

At Bellevue Hospital tepid sponges are used to reduce the temperature, the water being from 80° to 85°.

At Roosevelt Hospital alcohol sponges or "anterior packs" are used. I made inquiries to ascertain if tub baths are given in pneumonia in the various hospitals, and found that Bellevue and Roosevelt never use them; the New York very seldom, and only in cases with toxemic pyrexia. At the Presbyterian they are used in alcoholic cases when nervous symptoms are present.

Cough—When cough is a distressing symptom, all the hospitals give codeine. Ordinarily it is the only drug they use. When it is commenced it is given in regular doses of one-quarter of a grain every four hours. All the hospitals, with the excep-

tion of Bellevue Hospital, at times use heroin in one-twelfth-grain doses. The third division of Bellevue prefers morphine. At times Roosevelt Hospital gives morphine to aid a distressing cough.

Insomnia—For insomnia, which is often a distressing symptom, the three drugs most used are trional ten grains, veronal ten grains, codeine one-quarter of a grain. The Presbyterian uses any one of these indiscriminately. The New York and Bellevue generally use trional. Roosevelt only veronal and codeine.

Pulmonary Edema—Pulmonary edema is treated at the Presbyterian and Bellevue Hospitals by hypodermic injections of adrenalin; fifteen to thirty minims of one in a thousand solution is given every fifteen minutes, for five doses, then every half-hour for four doses. Atropine hypodermically, one one-hundredth of a grain, is also used at these hospitals. They all cup the chest for pulmonary edema; however, at the New York and Bellevue Hospitals greater reliance is placed upon increased stimulation. Oxygen is not given as much as formerly in any of the hospitals. It is only used in cases of dyspnea and cyanosis. The oxygen tank seems to still hold its place as the actual forerunner of the end.

Cardiac Failure—As we all know, the critical period in a case of pneumonia begins with the evidence of cardiac failure. The first indication of systolic weakness and failure of arterial tonicity leads to the administration of cardiac stimulants. The three cardiac stimulants which are used in all the hospitals are alcohol, strychnine, and digitalis. I find the use of these drugs varies a good deal in the different institutions.

(*a*) *Alcohol*—The cardiac stimulant which occupies first place and upon which the greatest reliance is placed is alcohol. Whiskey is the form used, in doses of half an ounce, the time of its administration varying from every hour to every four hours, according to indications. These indications are a small, compressible, rapid, feeble, and often irregular or intermittent pulse, associated with the absence of the first sound and a diminution in the apex beat.

At the Presbyterian Hospital alcohol is not commenced until there are *absolute* indications, such as cardiac insufficiency and marked constitutional symptoms—nervous symptoms, dryness of the tongue, etc. I find that alcohol is less used in this hospital than in any of the other institutions. At the New York Hospital

it is used especially in alcoholic pneumonias. Inquiries at the different hospitals show, with the exception of the Presbyterian, that alcohol is used to-day as much in pneumonia as it was two or three years ago. It is used at the Presbyterian less generally than it was.

(*b*) *Strychnine*—There has been a peculiar thing about the use of the second drug in pneumonia—strychnine. Most of us can remember when we first began the practice of medicine that strychnine was seldom, if ever, given as a cardiac stimulant in pneumonia; now it is universally used. Clinical experience has confirmed what physiological experimentation has long ago proven. When we remember the physiological action of this drug, how it stimulates the heart and tones up the arterial system by its direct stimulant action on the vasomotor centers, and not like alcohol and digitalis by its action on the heart muscle and the arterial wall, we find an explanation of its effectiveness; it whips up the heart, one might say, from its powerful nerve centre. Strychnine is never followed by the secondary depression so often seen after alcohol. I find this drug universally used in the hospitals. In talking over its use with the various house physicians, I learn that the two indications which call for its use are: (1) When whiskey is not able to hold the heart they always use it; however, secondary to whiskey—to re-enforce it, as it were. (2) When there is evidence of pulmonary edema and cyanosis: here it is generally given hypodermically in 1/20-grain doses. At two hospitals this second symptom is the special indication for its use. All alcoholic cases were especially found to respond to the action of this drug.

(*c*) *Digitalis*—At none of the hospitals, with the exception of Roosevelt, is digitalis used to any extent. I understand from the house staff at Roosevelt Hospital that two of the visiting physicians use it quite extensively when there is evidence of cardiac dilatation. It is used very rarely at Bellevue on the *second* or *third* division; on the *first* when there is evidence of pulmonary edema, and on the *fourth* when there is marked failure of compensation. At the Presbyterian Hospital digitalis is only used when there is evidence of cardiac dilatation or irregular action. In all the hospitals the tincture is the form in which digitalis is almost generally given, the dose being from five to ten minims every four hours. Fluid extract in two-minim doses is sometimes used at Bellevue and New York Hospitals.

(*d*) *Nitroglycerin*—The only other drug I found used in the hospitals is nitroglycerin, and this only very occasionally. At the Presbyterian it is never used, rarely at the New York Hospital, and only at the Roosevelt in the service of one of the visiting physicians. At the Bellevue Hospital it is more generally used, especially on the fourth division. The dose is one-fiftieth of a grain, generally given hypodermically.

I find that during the past year or so a number of drugs have been tried in pneumonia and discarded—for example, iodide of potash, sodium salicylate, pneumococcus serum, and creosote, although I may say the latter drug is still sometimes used by the different institutions, especially at the Presbyterian and Roosevelt Hospitals. The staffs of these two institutions inform me they can see no results from its use. At Roosevelt Hospital they are using saline injections into the rectum, especially in alcoholic cases and in cases in which nutrition is failing. Eight ounces of a normal saline solution is introduced into the rectum through a funnel attached to the end of a catheter. These saline injections are given about once in four hours, are well retained, and the staff informs me in many cases marked beneficial results are obtained. I should think this treatment would warrant more extensive use.

Before closing, I would like to make three statements in reference to the treatment of pneumonia, which clinical experience warrants me in considering of some value:

First, a plea for the more general use of morphine hypodermically in the early stage of the invasion, a stage in very many cases accompanied by not only the shock to the nervous system from a sudden and overwhelming toxemia, but the distressing pain often amounting to agony of a pleurisy, associated with the development of the pneumonic processes. The shock to the nervous system in many cases is intense. Patients generally rally from this condition, but often with a marked cardiac weakness. I believe if we made a routine practice of giving cases presenting these symptoms one or two full doses of morphine hypodermically, we would not only relieve the pain but would minimize the nervous shock, and certainly, remembering how morphine sustains the heart, we will start our patient on the course of the disease in a very much better condition. Morphine later in the disease is but seldom required, and in my opinion must only be used then with the greatest care.

Secondly, if any criticism can be offered of our present treatment of pneumonia during the past few years, it is along the line of injudicious and often unwarranted use of alcohol. I have often questioned whether if we did away with alcohol entirely the mortality would not remain about the same, and possibly a great improvement be noticed. During the last two years, in my hospital service at Bellevue and New York Hospitals, I have given very little alcohol, not one-twentieth of the amount I gave formerly, and I feel that the results obtained warrant me in cutting its use down still more. The ordinary cases certainly, it seems to me, do better without it. My experience has been that in the intense alcoholic cases strychnine hypodermically gives very much better results than alcohol, and these are the class of cases in which alcohol is usually given in very large quantities. We all know in the various institutions for the cure of alcoholism how on the sudden withdrawal of alcohol they hold the heart by hypodermics of strychnine, even in the most desperate cases. An exceedingly good cardiac stimulant which I found to take the place of alcohol in pneumonia is the old-fashioned spirit of Mindererus, "liquor ammonii acetatis." I have been surprised to find how universal is the use of this drug in pneumonia by the old country practitioners. My attention was first called to its use at a meeting of a county medical society in western New Jersey a few years ago. The drug, as we all know, is a good diuretic and diaphoretic, affects temperature, and acts as a mild stimulant. I claim no specific action for it, but if it does not do any good it will do no harm, and I would like to bring it to the attention of the members of this society.

Thirdly, the last point which I would make, and possibly the most important, is that more of our patients are damaged than helped by the promiscuous drugging which is still too prevalent. I believe if fewer drugs were given in pneumonia a very much larger number of uncomplicated cases would recover with no medical treatment beyond an occasional laxative. The mortality to-day in the various hospitals seems to me unusually high. In the four New York hospitals which I have investigated, the mortality varies from thirty-five to forty per cent. At the Presbyterian Hospital, in six hundred cases, the mortality was 34.8 per cent.; at the Roosevelt Hospital, from 1903 to 1905, the mortality was about 40 per cent.; at the New York Hospital, from 1893 to 1894, 38 per cent.

SOME PROBLEMS IN THE TREATMENT OF PNEUMONIA.*

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In considering the problems involved in the treatment of acute croupous pneumonia, the physician's individual conception of the nature of the disease, the cause of the different clinical phenomena, and how these causal factors are to be themselves controlled, or their effects on the different structures of the body to be antidoted or mitigated, will dominate his treatment. If he has a well-defined belief and the courage of his convictions, his plan of treatment will be more or less definite. If he has no definite idea of what is to be accomplished by his treatment, he will try any and every plan proposed; he is always striving to find in the text-books or monographs some hard-and-fast scheme, and he readily accepts the one which is presented most dogmatically, only to discard it when a more positive writer presents a new one. There is at the present time no orthodox plan for treating pneumonia. We have never known as much as we do at the present time of the cause of the disease, its pathological course, its diagnosis, the danger signals—yet we bemoan the fact that, as in days gone by, its mortality is the opprobrium of scientific and clinical medicine, although pharmacology and bacteriology have given us so many weapons to combat the other acute diseases. Pneumonia will always claim a certain percentage of victims; some will get well without treatment, or in spite of treatment used; but there is a considerable number in which recovery will depend upon the treatment.

In judging of the value of any plan of treatment, it must be remembered that pneumonia varies according to severity and type in different years, at different times of the year, and in different localities; that those who are affected are individuals differing widely in mode of living, constitution, and natural powers of resistance; that a plan of treatment suitable in one epidemic, or

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one locality, or for one class of people, is not always applicable, but must be modified to meet the individual case. The physician must decide on the plan of treatment, what remedies are to be used, and for how long. It all depends upon his medical judgment. The surgeon can lay down the principles and give all the details of a surgical operation, but in the treatment of such a complex disease as pneumonia this is impossible. There is so much difference of opinion on essential points, so much confusion as to the means to be employed, and such conflicting statements as to the value of certain special plans, that a paper on treatment is a discouraging task.

I feel almost like apologizing for introducing the subject again, especially as I have no antidote, or sure cure, to present. I can merely give you my viewpoint of some of the problems involved in the treatment. As long as pneumonia was considered merely an inflammation of the lungs, the principles governing its treatment were comparatively simple. They were those that were applicable to all inflammatory conditions. The fever was the index of the severity of the disease and one of its chief dangers, and every effort was made to control it. It is needless to recount the means used. Many of them still persist in the treatment to this day, and, although the original reason for their use was long ago shown to be erroneous, still clinical experience deems them to be of value. As long as attention was centered on the pathological changes occurring in the lung in the course of pneumonia, the restriction of respiratory surface and the mechanical obstruction to the pulmonary circulation caused by the consolidated lung were considered the chief determining factors in causing respiratory and cardiac failure, and active treatment, both external and internal, was used to arrest or limit the morbid process, to check the congestions and incipient exudates, to diminish the coagulability of the blood, and, when consolidation had taken place, to hasten the absorption of the fibrinous exudate, and so overcome the obstruction. Although at the present time we know that the changes in the lung are but incidents of the disease, and that the mechanical obstruction caused by the consolidated lung is only a contributing factor in the production of respiratory and cardiac failure, certain measures that were found beneficial when pneumonia was considered a local disease still remain suitable treatment for the serious complications that occur during the cause of the disease, and it is necessary that we

study anew their mode of action and how they do good. At the present time pneumonia is considered an acute infectious disease, with a localized inflammation of the lung of a characteristic nature, due to the presence of the pneumococcus. The most difficult problem in each individual case is the determination to what extent the clinical phenomena of the disease are dependent on the toxemia on the one hand, and the pathological condition of the lung on the other.

How, in the light of our present knowledge, can we explain the sequence of events in mild, severe, and fatal cases? In mild cases the sudden onset, the rapidly rising temperature, and, before any recognizable change in the lung occurs, the increased respiratory rate, the greater depth of respiration, the bringing into use the accessory muscles of respiration, the dilation of the nostrils with inspiration, the almost diagnostic expiratory grunt, are pathognomonic. The circulatory phenomena are almost as marked. The cardiac action is forcible; the first sound booming; the pulse full and bounding; the systolic blood pressure high the diastolic also increased. The patient is restless, even when there is no pain. There is sleeplessness, delirium, and in children convulsions. In mild cases and in strong, robust individuals these "sthenic" symptoms may increase in degree throughout the entire attack, until they suddenly cease with crisis.

In severe cases the "sthenic" symptoms continue for a few days only. On the third, fourth, or fifth day according to the type, the respirations become more rapid, but shallower, cyanosis is present, and there develop gradually or suddenly all the symptoms of respiratory insufficiency. Cardiac action becomes more rapid, but less forcible, the pulmonic second sound is accentuated, the pulse loses its bounding character, the tension falls, and the pulse later becomes dicrotic and irregular. The systolic blood pressure falls, but earlier and more marked is the decrease in diastolic pressure. The entire venous system shows engorgement, and there are signs of overfilling of the right heart. The patient is apathetic, the face cyanotic, and the entire body covered with a cold, clammy perspiration. When this stage is reached, if crisis occurs, the temperature falls, symptoms of impending respiratory and cardio-vascular failure disappear, cyanosis clears up, and the danger is passed.

In other cases, before the time of crisis comes, the patient dies of pulmonary edema, respiratory insufficiency, exhaustion of the

respiratory muscles, or cardiac failure, and at autopsy the cavities of the right heart are found dilated and filled with clots, which may be partly decolorized and extending into the pulmonary arteries. The left ventricle is often found firmly contracted and empty, less frequently dilated and filled with recent clot.

How far are the dangerous symptoms due to curtailment of respiratory surface and mechanical obstruction to the circulation? In severe cases with recovery the symptoms of respiratory insufficiency and impending circulatory failure rapidly disappear with the crisis, although there is no change in the extent of lung involved, the degree of consolidation, or obstruction to the pulmonary circulation. In other diseases of the lung and pleura that diminish to a greater degree the respiratory surface or produce more marked obstruction to the pulmonary circulation, when the patient is quiet there is not present the rapid labored respiration or the effect on the pulmonary circulation and right heart, even though there be high temperature. The local lesion in the lung is therefore not the essential cause of the symptoms, although it contributes to the seriousness of the case and adds its own peculiar danger.

The toxemia of the pneumococcus infection must be considered the chief factor in causing the symptoms of the disease. Theoretically, this is conceded by nearly everybody, but, therapeutically, it is ignored in practice. Professor Jurgensen says: "If pneumonia is an infectious disease, it must be treated according to the same fundamental principles which experience has proven valuable in other infections." As a therapeutic maxim, the statement of Professor Jurgensen is comprehensive, but to treat the disease intelligently, it is necessary that we know what is the nature of the action of the toxemia on the different organs of the body. Is it a stimulant, an irritant, or a depressant? Does it act on one organ or system of organs in one manner and on others in an opposite? Are its early and late effects of a different nature, or are the late effects the result of the early ones? Upon our opinion will depend the general management and the choice of remedies to control the various symptoms and complications that arise at different periods of the disease.

I believe the primary and essential action of the toxemia of the pneumococcus infection is that of stimulation. Its effect upon the general nervous system is shown by the restlessness and sleeplessness, and in children by convulsions. The respiratory symp-

toms that occur with the onset of the disease, the rapid, deep breathing, the using of the accessory muscles in both inspiration and expiration, the fuller expansion of the thorax, the intensified respiratory murmur over the unaffected portion of the lung, indicate that the entire respiratory mechanism is being stimulated. The respiratory phenomena cannot be due to fever alone, for they do not correspond to the degree of temperature, and in other febrile diseases they are absent, or present in a much less degree. They cannot be attributed to the pain alone, because they occur even when pain is absent; nor dependent upon restriction of respiratory space, for they occur before there is much restriction and disappear with the crisis, although the same amount of pulmonary involvement exists as when the respirations were so much disturbed. The fever, pain and pulmonary consolidation only add their quota to the respiratory symptom, although later they may be determining or contributing factors in producing respiratory failures. The cardiovascular system shows like signs of stimulation. The rapid and overforcible action of the heart, the full, bounding pulse, the high systolic blood pressure, all show that the functional activity of the heart is increased.

The above respiratory and circulatory phenomena present during the early period of the disease may continue throughout, increasing in intensity as far as the symptoms of stimulation are concerned, and never showing any signs of respiratory or circulatory failure; but in most cases, sooner or later, according to the severity of the disease, the age, habits, previous physical condition or constitution of the patient, there appear gradually or suddenly the signs of either general weakness or insufficiency of some organs, or system of organs, most frequently respiratory, or circulatory, or both. The respirations become more rapid, but shallower. They lose their full, deep character, the respiratory muscles contract spasmodically but with less effect, the amount of tidal air is markedly reduced, there is imperfect ventilation of the alveoli of the unaffected portion of the lung, and cyanosis develops. The cardiovascular changes are, increased rapidity of the heart (120 or more), the pulse becomes short, dicrotic, and of low tension; there is evidence of imperfect filling of the arteries and accumulation of the blood on the venous side, with overfilling of the right heart, and engorgement of the pulmonary circulation. To what are we to ascribe these changes? Is the nature of the action of the toxemia changed? Is there now a depressant

or paralyzing action on certain structures, which action was masked by early stimulation and the effect of fever, pain, and pulmonary involvement? We know that in infectious diseases, according to the degree of the toxemia and length of time it has been active, there are parenchymatous changes in all organs, and especially in the heart muscle. In fatal cases of pneumonia this is often present. Yet in a considerable percentage of cases it is not sufficient to account for the degree of cardiac insufficiency noted during the latter period of the disease.

I believe the stimulation of the early period of the disease is accountable for the later failure—that it is a true exhaustion that follows the excessive work that the respiratory and circulatory system has had to do. We have its analogy in the exhaustion that follows toxic drug stimulation in the pharmacological laboratory. I never see a case of pneumonia, but I wonder whether he can stand the pace, for he is in an endurance race against time, the time of the crisis. If the pace has been rapid from the start; if he is handicapped by age; if his resisting powers have been impaired by work, worry, bad surroundings, or improper or insufficient food; if his nervous system has been rendered unstable by alcoholic or other excesses; if he is suffering from structural disease, the chances are against him. Proper treatment alone is able to aid him, and it will do the most good the earlier in the disease it is given.

If the pneumotoxin has the action I have described, then there are definite indications for treatment at every stage of the disease. From the very onset our efforts should be directed to controlling the toxemia and its effects: (1) By limiting the production of the toxin in the lung and elsewhere; (2) by antidoting it after its absorption into the blood; (3) by diminishing its amount through free elimination, and (4) by neutralizing or combating its effect on the organism.

As the pneumotoxin is chiefly produced in the affected area of the lung, numerous treatments have been proposed to limit the extent of the pulmonary involvement. To control the pulmonary congestion abstraction of blood by venesection, leeching or wet cups, withdrawal of blood from the lung to the surface of the body by dilatation of the superficial blood vessels through counterirritation, hot applications, etc., or the action on the inflamed area, by the ice bag, cold coil, or cold pack, have been advised. Each method has its advocates, who believe that their

plan is efficient, or even curative. All these means have a use, and, in proper cases, do give relief to the patient. They often seem to influence the circulation of the unaffected portion of the lung, and possibly in this manner prevent more widespread involvement; but beyond this effect I have never been able to see that they affected the inflamed area itself, or hastened its resolution.

To act directly on the pneumococcus in the lung, either by destroying it, diminishing its vitality, or rendering it less virulent, or by antidoting the pneumotoxin, creosote for its compounds, both by inhalation and internally, sodium salicylate, carbolic acid, salol, quinine, and other members of the antiseptic group have been advocated. Specific action has been claimed, and histories and statistics have been published to prove their value. The advocacy of these remedies by trustworthy observers, in whose judgment we all have confidence, should cause us not to ignore their possible value. At one time or another I have given them all a thorough trial. I have never seen any specific action, but in special cases, given at the right time, I am convinced that they often exert a favorable influence upon the disease. Time and again I have seen irreparable harm done by their injudicious administration or too long continuance. If they are to be of service, or to be administered with safety, it is during the early stage of the disease. When the symptoms of cardiac and respiratory exhaustion are present their use is contraindicated. The time has then gone by when they can be given with safety. Any possible effect they can have on the pneumococcus, or the pneumotoxin, is more than counterbalanced by their depressing action on the general nervous system and other organs. Quinine is an exception to the rule. Given in moderate doses, even late in the disease, it does not produce any depressing action. But I have never seen such definite good effects from it that I consider it of use. In judging of the value of these drugs in controlling the disease, the effect on the temperature, or the fact that the disease more often terminates by lysis than by crisis must not be taken as a guide. Given in sufficient doses, they will reduce the temperature; but unless, with the fall of temperature, there is a corresponding improvement in the nervous, cardiac, and respiratory symptoms, they are not benefiting the patient. Sthenic cases bear them better than the aged, the debilitated, and the alcoholic.

With the already known fact that pneumonia was a self-limiting disease, the discovery that the pneumococcus was the infec-

tious agent and encouraged by the success that attends the treatment of other infectious diseases, especially diphtheria and tetanus, with antitoxins we were in high hopes that an antitoxin would be produced that would be as efficient in this disease. Up to the present time no such serum has been produced. All the sera thus far introduced have not been antitoxic or antifectious enough to justify their use, although some writers claim that they have seen good effects.

As at the present time we have no proven means for preventing or controlling the production and absorption of the pneumotoxin or antidoting its action after it had been absorbed, our chief effort must be directed towards the elimination of the toxin as rapidly as possible and combating its effect on the different organs We have no definite knowledge of the avenues by which the toxins of pneumonia are eliminated, nor the form in which they leave the body. We are therefore compelled to use the means that have been found most efficient in other acute and chronic toxemias; namely, catharsis, diaphoresis, and diuresis. All these means should be judiciously used from the very beginning. They should not exhaust the patient. The using of calomel and a saline cathartic at the first visit is the orthodox method of procedure, but after this initial dose too little attention is given to the eliminative power of the intestines. As long as the bowels act once a day, it is deemed sufficient. Unless there is some counterindication, I use saline cathartics freely during the early days of the disease. Sweating should be produced by external means if the toxemia is urgent, and the same means employed as in the treatment of acute uremia; but as the hot packs, or hot-air baths, are somewhat depressing, active diaphoresis is not indicated, unless the toxemia is very profound, and it is impossible to get sufficient action of the kidneys. Pilocarpin should not be used, as it is extremely dangerous in pneumonia. On the other hand, the free use of liquor ammoni acetatis citrate of potassium, and hot drinks, especially hot water, are useful.

The chief reliance is on elimination by the kidneys. For this purpose water, either plain or aerated, should be given plentifully. In nearly every case of pneumonia the urine shows some albumen, indicating that there is kidney irritation—probably due to the action of the toxins—and dilution of the urine is called for. Usually the urine is decreased, high colored, and concentrated. The rapid, deep breathing and the free perspiration that are present cause a larger amount of water to be eliminated by other chan-

nels, and, unless an extra quantity of water is given, the urine decreases. On account of the presence of nausea and vomiting in some cases, and in others the tendency of the stomach to retain the fluids given, a free administration of water by mouth is prevented. Under such circumstances there should be given, three or four times a day, enemas of normal saline solution (drink enemas). Water is very rapidly absorbed from the bowels, and is followed by free diuresis. In the early period the stimulating diuretics should not be given. The alkaline mixtures, as the A. B. C. diuretic, citrate of potassium, cream of tartar lemonade (which is a very agreeable and refreshing drink), liquor ammoniæ aetatis (half ounce doses every two, three, or four hours) are the most serviceable. Drinking water as hot as can be taken is well borne by many cases in which nausea and vomiting are present. Many cases suffer from lack of water, because of the fear that if water is given the patient will not be able to take sufficient nourishment. It is better to give the needed water, as most patients are overfed. Anyone who has personally gone through an attack of pneumonia and has had milk, broths, etc., given *ad nauseam*, when what he has craved most was a good drink of water, will appreciate the hardship. Later in the disease, when there is circulatory failure, the use of diuretics of the theobromine group may be used to meet indications.

In those cases in which the patient seems to be overstimulated by the toxemia, when the cardiac action is excessive, the pulse very full and of high tension, or where the kidneys are affected and the urine highly colored, venesection is indicated, the amount of blood withdrawn being governed by the effect. Enough should be taken to lower the tension of the pulse and quiet the heart. If there is fear of reducing the blood mass too much, normal saline may be introduced into the veins, or given by hypodermoclysis. There are two periods when venesection is indicated: early in the disease for the toxemia, and later, when its purpose is to reduce venous stasis.

It has been claimed that oxygen, when given systematically throughout the disease, has the power of diminishing the toxemia. We have already heard to-night of the value of fresh air. Formerly, through the mistaken idea that a pneumonia patient should be kept as warm as possible, all air was excluded from the room. The treatment of other infectious diseases by the fresh-air method has overcome popular prejudice. I fear, however, we are in danger of bringing a very valuable plan of treatment

into disrepute by overdoing the matter in pneumonia. Fresh air is needed, but low temperature is not beneficial. I do not think the temperature of the sick room should fall below 60° F. I generally try to keep it about 65° F. Experience has shown that, whenever tuberculous patients who are taking the open-air treatment contract pneumonia, they do best when protected against low temperature. Cold is not an essential element of the open-air treatment.

"As we cannot cure our patient, we must endeavor to keep him alive until he cures himself." To do this it is necessary to combat the effects of toxemia on different organs of the body. The treatment will, of necessity, be symptomatic, but it is none the less rational. All agree that the powers of the patient should be conserved, and, when the powers are failing, should be assisted. But there is great difference of opinion as to how this can best be done. Believing that the grave conditions that occur in the late days of the disease—the respiratory insufficiency and cardiac failure—are, in the major part, due to the exhaustion of these systems due to the excessive functional activity caused by the stimulation of the toxemia, I consider that at the very beginning we should try to abate this overwork. Granting that the symptoms are those of stimulation, what should be our guide for the administration of medicine to control it? Does it affect chiefly the central nervous system, or does it also have some action over the neuromuscular mechanism of the heart and lungs? It is impossible to answer many of these questions definitely, or to fortify our conclusions with experimental data. I believe that the action of the pneumotoxin is a widespread one and shows, just as other poisons do, an individual variation in its effects on the nervous system and organs. Generally the ushering-in symptoms give indication as to what portion is most susceptible to the toxemia. In the majority of cases the chill, restlessness, characteristic insomnia, cardiac and respiratory symptoms show that the nervous system, and especially the medullary centres, are acted upon. There is a great difference of opinion as to whether or not we should actively combat the above symptoms. Personally, I believe that they should be controlled, using such means as are indicated in individual cases. Rest in every sense of the word is necessary in all cases. Good nursing and proper surroundings are conducive to this. To the head, cold compresses or ice bags, and over the chest cold compresses, as recommended by Dr. Baruch, have a sedative action on the nervous system, and act

favorably upon the heart and respiratory muscles as well. I believe most of the good effects that have been ascribed to the ice bag in controlling pneumonia are due to the action of cold on the heart rate. There are cases in which it is necessary to use the bath treatment as fearlessly as in typhoid fever. Fortunately, these cases are rare. The indications are not so much the high temperature as the condition of the nervous system, where the toxemia seems to be overpowering it. In this condition the high temperature is a part, but not the cause of the symptoms.

When quiet, good nursing, eliminative and simple hydropathic measures fail to control the restlessness, insomnia, delirium, and other nervous symptoms; when there is pain and harassing cough, should active treatment be used? I believe that sleep and freedom from pain should be procured as early as possible, and the patient kept quiet and comfortable. When there is pain, opium or one of its alkaloids generally acts best. The form to be used will depend on the case. Morphia, hypodermically, is best suited for the initial dose. Frequently the statement is made that morphia, while relieving the pain, has made the patient more nervous and has accelerated the heart. The practice of combining atropia with morphia is, in the majority of instances, accountable for this. In most cases in which morphine is given for relief of pain, the action on the medullary centres is not desired, and so atropine is given to antagonize it. In pneumonia this antagonistic action is not wanted. The respiratory centre and heart are, in pneumonia, especially susceptible to the action of atropine; it is therefore contraindicated in the early stages of the disease. There are individuals whom opium and morphine render restless and semi-delirious whenever given; if this idiosyncrasy is known opium should not be used. When opium does not act well, or when pain is not present, the bromides, or chloral, either singly or in combination, or the synthetic hypnotics, may be used to induce sleep. In the early days of the disease I have never seen these drugs produce any bad effects. When rest and sleep are obtained at the beginning, much less medication is needed later to quiet the patient. With the control of the nervous symptoms there is generally improvement in the respiration; it is less rapid. If full expansion of the thorax has been prevented by pain, the breathing becomes easier, cyanosis disappears, providing the medicine has not been pushed too far. It is especially important to relieve pain, when it prevents the patient from breathing easily, except when in one position. When there is no pain, but the patient is breath-

ing very rapidly, although there is no evidence of imperfect oxygenization, heroin is useful. In these cases the rapid respiration is apparently due to increased irritability of the respiratory centre.

In the alcoholic, and certain non-alcoholic patients the restlessness and insomnia can only be controlled by the free use of alcohol. Its action in these cases seems to be chiefly sedative.

It is always a debatable question whether any cardiac sedative should be given, when at the very onset there is a rapid overacting heart, bounding pulse, and high systolic blood pressure. The ice bag, cold coil, and cold pack give some relief, but in many of the cases do not control sufficiently. I have watched the course of cases under the care of physicians who use the old "antiphlogistic" plan of two decades ago, who at the onset of the disease give aconite, vertatrum viride, and even tartar emetic, and have been surprised to see the controlling effect on the heart. Their plan is to give the sedative during the first eighteen or twenty-four hours, in sufficient doses to control the heart, and then in smaller doses at longer intervals for two, three, or four days, watching carefully the pulse, and decreasing or stopping the drug as soon as the tension falls below a certain point or there is any suspicion of dicrotism. When given in this manner, I have seen no bad effects or any late symptoms that could be ascribed to the drug. I have never had the courage to use either veratrum viride or tartar emetic. The older practitioners have gained a knowledge of the action of these drugs, which comes only with longer acquaintance. I do use aconite in those cases where the cardiac rate and power are out of proportion to the other symptoms. I rarely continue it beyond the first eighteen or twenty-four hours. The usual dose is five to ten minims (2-3 minims Old Pharmacopeia) every two or three hours. I believe aconite is much safer to use than we have lately been led to believe.

Patients with pneumonia die most frequently from cardiac failure, respiratory insufficiency, and pulmonary edema. All three are generally present in the terminal stage. In pneumonia we must be on the watch for the first symptoms of weakness, and success in treatment will largely depend on our ability to prognosticate in each case at what point the failure is likely to occur, and on our power to combat or control it at the start before exhaustion at one point throws an added burden elsewhere and causes a general failure, when all treatment is useless. Usually the first evidence is shown by the cardiovascular system. The

heart becomes more rapid, the pulse small, short and dicrotic, the systolic blood pressure falls, there is imperfect filling of the arterial system and overfilling of the venous with dilatation of the right heart and engorgement of the pulmonary circulation. The remedies used to combat these symptoms will depend on our conception of the causes of the failure. That so many diametrically opposed plans have been advocated shows that there is a great difference of opinion. The primary failure may be caused by changes in the heart muscle, or by changes in the vasomotor control over the blood vessels, leading to vascular collapse. It has long been known that the toxin of pneumonia does not cause as marked weakness of the left ventricle as of the right; that, in addition to the greater parenchymatous degeneration of the right heart we have the added burden of the interference with the pulmonary circulation by the consolidated lung. This mechanical obstruction has been generally considered the exciting and chief cause of cardiac failure.

Romberg, in considering the effects on the circulation of pneumococcus infection, says: "We must consider with the heart weakness the weakness of the vasomotors; with the cardiac paralysis that of the vasomotor also." I am convinced that to the loss of vasomotor control must be ascribed the chief danger in pneumonia. I think this gives us a guide for treatment, and that it harmonizes many discordant opinions, concerning the value of certain drugs as cardiovascular stimulants. When there is loss of resistance in the arteries and arterioles producing marked disturbance of the equilibrium of the circulation, although the left ventricle is relatively strong, the pulse becomes more rapid, small, and dicrotic. The blood passes rapidly to the venous side, causing an incomplete filling of the arteries, while the veins become overfilled. The increased pressure in the veins, slight though it be, and the rapid respirations tend to cause the right auricle to be distended. The weakened right ventricle cannot empty itself against the increased pressure present in the pulmonary circulation, and there develops right-side cardiac dilatation and insufficiency with tendency to cardiac thrombosis, and later pulmonary edema and respiratory failure. To combat this condition nitroglycerin is frequently advised, under the belief that by dilating the arteries the work of the heart is decreased. But peripheral resistance is already too low on the arterial side, and nitroglycerin only makes it lower. We have no data to prove that nitroglycerin has any effect on the pulmonary circulation. Personally, have

only seen bad effects when nitroglycerin and other members of the nitrite group are given in pneumonia during the stage of vasomotor weakness. It is only indicated in pneumonia when there is high arterial tension associated with a rapid-acting heart, as occurs in cases of arteriosclerosis and nephritis. Under such conditions it should be given from the very beginning, as the rapid and forcible acting left ventricle soon exhausts itself pumping against high blood pressure. I cannot too strongly condemn the tendency of resorting to nitroglycerin whenever cardiac failure is suspected.

Against the loss of vasomotor control we have two classes of stimulants. (1) Those acting on the medullary centres—strychnine, caffeine, atropine and cocaine. (2) Those acting directly on the muscular tissue of the arterial system—digitalis, ergot and suprarenal extract. When loss of vasomotor control is associated with evidence of weakness and exhaustion of the other medullary centres, as rapid, shallow respirations and cyanosis, marked apathy of the patient, strychnine should be the stimulant selected. The dose and frequency of administration should be just sufficient to increase the irritability of the centres. If carried beyond this point, the overstimulation only hastens the time of absolute failure. I know of no drug that is able to do so much good and also so much harm in pneumonia, as strychnine, and the deceptive feature about it is that under its free use the patient's condition improves and the secondary result of overstimulation is lost sight of. Strychnine should not be a routine treatment in pneumonia. It should not be given until indications for its use are present. As I said before, a patient suffering from pneumonia is in an endurance race against the time of the crisis. The spur should not be used too early or too freely. He may need all the stimulation possible at the end. Atropine, cocaine, and caffeine are stimulants that can be used for the emergencies that occur at the crisis, although when strychnine has lost its power, we are generally leading a forlorn hope. In cases of respiratory failure both atropine and cocaine have the power to stimulate the respiratory center. I prefer cocaine to atropine, and when it is applied to the inner side of the nostrils, either as a spray or by painting with a camel's hair brush, it has a maximum effect on the respiratory centre, as I have frequently seen in treating respiratory failure caused by morphine poisoning. When the vasomotor centre does not respond to strychnine, or when the vasomotor loss of control occurs before the weakness of the other

centres, and the giving of strychnine would unduly stimulate these centres, the blood vessels may be acted on by digitalis, ergot, or suprarenal extract. The value of digitalis in treating vascular collapse in pneumonia is a very decided one, and there is no drug that can take its place. It is a well-known clinical fact that persons whose normal blood pressure is very low do not bear pneumonia well. The cardiac rate is very rapid, the pulse dicrotic and right side cardiac dilatation occurs early. The judicious use of digitalis in these cases during the early stage, combined with aconite, slows the heart and restores the vascular tone. Later in the disease digitalis alone is indicated. When there are added symptoms of exhaustion of the other medullary centres also, strychnine may be used in combination with digitalis. Digitalis should be given in pneumonia for a definite purpose. The objection to its use has been due to the giving of large doses throughout the disease, on the theory that it has some specific action. The power of ergot to contract the arteries is conceded by all. Whether this effect is due to vasomotor stimulation alone, or to peripheral action also is undecided. The fact that the physiological action of ergot is so definitely restricted to the vasomotor centre and the blood vessels causes it to be especially valuable in cases of pneumonia with low arterial tension. In alcoholic cases the long-continued use of alcohol, provided it has not induced arteriosclerosis or nephritis, has produced a depression of the vasomotor centre; the capillary circulation is also abnormal. In pneumonia, in these cases, there is a peculiar lividity of the surface that is not due to cyanosis from respiratory insufficiency. The pulse has a very low tension, and the sputum is hemorrhagic and dark, not the ordinary brick dust type. Ergot, by restoring the vascular tone, meets the most urgent symptoms. In many cases of pneumonia in the alcoholic, strychnine, while improving the circulation, increases the delirium and restlessness; it is necessary to give alcohol and narcotics in order to induce quiet and sleep, but these still further disturb the peripheral circulation. The use of ergot counteracts their effects on the circulation. It should be administered hypodermically, using the aseptic preparation.

Suprarenal extract or adrenalin chloride acts on the blood vessels very rapidly and energetically, but its effects last but a short time. Its use is to be reserved for the treatment of sudden vascular collapse, where we are using every effort to keep the patient alive in the hope that the powers of resistance may

finally overcome the disease. For no case of pneumonia is to be given up until absolutely dead. The diffusible stimulants—camphor, musk, aromatic spirits of ammonia, Hoffman's anodyne are all emergency remedies.

Time does not permit me to discuss some of the other problems. The rather active treatment I have outlined, especially for the early stage, will be criticised as contrary to the present treatment—the expectant plan. If the expectant plan is merely a treatment of observation, of standing idly by until the grave conditions of the disease develop, then what I have suggested is contrary to it. If, on the other hand, it means that we fully realize the dangers that are to be expected and try to ward them off, then I have only described what is to me a rational expectant treatment. The opportunity to conserve the patient's strength occurs early, and if this be the main object of our treatment, then the early stage of the disease is the stage in which the fearless and intelligent use of active measures will be conducive of the greatest good. And we should not neglect our opportunities and wait passively until conditions arise which either contraindicate or render useless their employment. For when this stage is once passed "all the rest is mere work of rescue and success is a question of vitality."

THE TREATMENT OF LA GRIPPE.*

By Charles E. Nammack, M.D.,

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There is neither a prophylactic nor a specific for influenza. It is obviously impossible to formulate a specific plan of treatment for an acute infectious disease, occurring in widely-extended epidemics, characterized by catarrhal inflammation of the mucous membrane of the entire respiratory tract, and less frequently of the digestive tract, and attended by quickly oncoming debility, and by nervous symptoms. Not only is the disease exceedingly variable and protean in its manifestations, but we must differentiate, in the individual instance, between a case occurring during a pandemic invasion, as an endemic incident, as an epidemic occurrence, or as a sporadic outbreak. In the first instance, the element of panic enters largely into the calculation. In the second, the environment must be considered. In the third the prevailing type of the epidemic must be estimated. In the fourth we must carefully differentiate the ordinary catarrhal respiratory inflammation, which has an obvious exciting cause—usually changeability of the weather, lacks the characteristic depression, does not attack many members of the same family, and has, as a rule, a lower range of temperature and less involvement of the nervous system. I will say nothing here about the necessity for excluding acute miliary tuberculosis, typhoid fever, malaria, or one of the ordinary infectious eruptive fevers.

Having decided that we have to deal with a case of general influenza due to Pfeiffer's bacillus, or probably to the residual germs which remain after a pandemic invasion, our first duty is to endeavor to prevent the spread of the disease. To this end, we should isolate the patient, even though we cannot reasonably hope, by this measure, to diminish either the activity of the infecting principle or the susceptibility of other individuals. The nasal and bronchial discharges should be disinfected with the

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same careful attention to detail that is shown in other acute infectious diseases. The patient himself should be at once put to bed, for, although prophylaxis against the attack is theoretical and to a great extent impracticable, prophylaxis against sequels is in the highest degree practical and useful. Those who continue at work and endure exposure not infrequently suffer at a later period of the disease from the most serious results. A fair-sized, single dose of calomel, followed if necessary by a saline draught, will help to eliminate some of the infection if given at the time of the initial chill. The patient's bed should be warmed, and he should receive, in the bed, a footbath of hot mustard water with rubbing of the extremities, and a hot drink. Alcohol will be useful in those accustomed to its use, also in the aged, and in the weak. Young, healthy, abstemious patients will not need it at this stage. The diet should be restricted to milk, albumen water, gruel, and the ordinary fever foods. The use of milk should not be pushed in those who have a heavily-coated tongue, for it will only cause indigestion. Concentrated meat broths can be used, unless active diarrhea or the condition of the kidneys should be a contraindication. Acidulated drinks will relieve the thirst in some, and also soothe the stomach, and flush the kidneys. Others will be benefited by alkaline fluids, such as vichy, or by bland waters. Cold baths and cold compresses are generally not well borne, and increase the discomfort.

The average patient will expect to swallow drugs, and the atrocious pains sometimes present will suggest the use of analgesic antipyretics. It is my personal conviction that drugs of this class contributed more to the death rate during 1889-1890 than did Pfeiffer's bacillus. At the present time their use is not so general, but they still occupy too prominent a place in the lay mind. Pain which is severe enough to require drugging will be better managed by Dover's powder or codeine. Another drug largely used by self-prescribers in this affection is quinine. There is not the slightest evidence that quinine in any dose favorably modifies the course of the attack, or shortens its duration, although it may be useful during convalescence. The old rule that "the physician is to be a watchman all the time and a therapeutist only when occasion arises" applies with striking force to influenza, yet the symptoms of this affection cry aloud for relief, and of these symptoms the depression and the respiratory catarrh will in most cases be prominent. I usually meet these symptomatic indications by a prescription containing five grains of ammonium

carbonate, five grains of ammonium salicylate, and half an ounce of the aromatic wine of erythroxylon coca (of the National Formulary) in each dose. This may seem like active therapy, but this combination has done me yeoman service during many years in private practice, and in dispensary and hospital services. It will relieve headache and neuralgia and stimulate the heart, respiration, and kidneys. Incidentally, I may remark that it is sometimes fearfully and wonderfully made by apothecaries who are pressed for time. When this combination fails to meet the indications mentioned, the case is serious enough to require constant attention, and the hypodermic use of strychnine, caffeine, or camphor. The stage of convalescence must be carefully managed by tonic and supporting treatment, especially addressed to the nervous system, in order to avoid the formidable array of complications and sequels, any of which may develop even in mild cases.

The most common complication is bronchitis. This will require its usual treatment, with the important reservation that no remedy should be employed which is a sedative to the nervous or circulatory system. This reservation excludes antimony, aconite, veratrum viride, potassium salts, and large doses of ipecac, and compels us to fall back upon steam inhalations, hot drinks, counterirritation, rest in bed, and codeine for excessive cough. Should bronchopneumonia develop, strychnine and aromatic spirits of ammonia, well diluted with water, are indicated. Oxygen inhalations, though much derided, are advantageous in some cases. The presence of profuse bronchial secretion in the smaller tubes positively contraindicates morphine.

The poison of influenza is capable of setting up serious changes in the intracardiac ganglia and in the myocardium; therefore, the pulse and the heart sounds must be carefully studied, and all unnecessary movements on the part of the patient absolutely prohibited until the rhythm and force of the heart have been restored. The three tinctures, nux vomica, belladonna, and digitalis, act well in bringing about this result, or a similar combination of strophanthus, cactus grandiflora, and belladonna, sometimes acts efficiently. The coexistence of valvular disease of the heart will add its own specific indication for treatment.

Kidney impairment will also supply a field for therapeutic intervention, which is too wide to be gone over within the limits of this paper. Symptoms referable to the nervous system may dominate the clinical picture from the onset, or may become prom-

inent at any time during its course. It is no uncommon sequel to see robust, self-reliant men converted into feeble hypochondriacal valetudinarians by an attack of grip, which has weakened the general nervous mechanism, as well as the special nervous apparatus of the heart. This sequence of events seems especially liable to happen to scholars and brain workers, who should receive the benefit of a long period of rest with entire change of scene and employment before taking up again their life work.

The tragic results of the formation of a drug habit during this period should make us extremely cautious in inaugurating the use of hypnotics, sedatives or analgesics. Headache, insomnia, neuralgia, neurasthenia, hysteria, chorea, peripheral neuritic disturbances, and psychic disorders, may all seem to the patient to demand drugs, but the practitioner who relies upon outdoor life, freedom from worry and care, moderate exercise within reasonable limits, good nerve-building diet, and hydrotherapy, will leave in his wake the more grateful memories. In the stubborn neuralgias during the period of convalescence, quinine, in moderate doses, is more effective than its modern coal-tar competitors, and is frequently useful where they have failed.

THE PSEUDOMALARIAL TYPES OF INFECTIVE ENDOCARDITIS.*

By Warren Coleman, M.D.

Cases of infective endocarditis simulating malarial fever have been reported from time to time for half a century, yet adequate recognition has scarcely been given to the subject in text-books. Nor has the attempt been made, so far as I am aware, to bring together the various types. The importance of infective endocarditis and the difficulty often encountered in arriving at a correct diagnosis apparently justify a consideration of the pseudomalarial forms under which it may be masked.

Before the discovery of the malarial parasite, especially, the attempt was made to establish an etiological relation between malarial fever and infective endocarditis. It was entirely *post hoc* reasoning. Convincing proof of such relationship has not yet been offered. In fact, it is almost universally conceded that antecedent or coincident malaria is merely fortuitous. Some cases have given histories of recent or old-recurring malarial attacks, but these histories must be accepted with caution, at least in certain instances. The pseudomalarial types of endocarditis are subject to complete, though temporary, interruption for weeks at a time. Herzog has reported the coincident presence of the malarial parasite in one case, but does not prove its etiological significance. It was present also in a fatal case treated at Bellevue, and in another at City (Charity) Hospital.

The cases of infective endocarditis of pseudomalarial type which have been reported fall broadly into two groups, acute and chronic. The duration of the acute cases is measured by weeks, of the chronic by months. It will often be found, however, that cases which have died after a few weeks' observation give histories extending over several months. Other chronic cases may continue for eighteen months or more.

The pseudomalarial must be distinguished from the frankly septic and pyæmic forms of infective endocarditis, of which they

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constitute a remarkable sub-group. They differ rather in the periodicity than in the nature of the paroxysms. Their resemblance to malarial fever is often very exact. The paroxysms may recur at regular periods, even at the same hour, and the intervals be fever-free. In the apyrexial periods the temperature is often subnormal. No special infective agent is responsible for the pseudomalarial types, nor is it necessary that there shall have been previous cardiac valvular disease.

The cause of the periodicity has not been determined. It has been thought to be due to disturbance of the heat-regulating mechanism by an antecedent malaria, and that the stamp of malaria is carried by the subsequent febrile movement. This seems unlikely, since no other febrile disease, when it follows a malarial attack, pursues such a course. Nor has it even been proved that the majority of cases have been recently preceded by malaria. Sicard found staphylococci in the blood in the pyrexial but not in the apyrexial periods of one case.

The following pseudomalarial types have been reported:

1. Double quotidian.
2. Quotidian.
3. Tertian.
4. Quartan.
5. Septan.
6. Mixed types.
7. Irregular.

A single case may present during its course several of these types, yet at a particular period each type be pure. It is almost characteristic of these cases that they change their course. In spite of this fact, it is advisable to speak of distinct types. Cases may be under observation for short periods only, during which a definite type is preserved.

When the disease is approaching a fatal termination, the paroxysms often return irregularly.

Characters of the Paroxysms—The paroxysms may be identical with those of malarial fever, presenting cold, hot, and sweating stages, with fever-free intervals. The cold stage may be accompanied by a prolonged, hard chill, or simply by sensations of chilliness. As in malaria, the temperature may rise steadily through the chill. The chills begin with or after the initial rise of temperature, which, though sharp, may occupy several hours

in reaching its maximum. In acute cases the maximum is often very high, even 107°. The defervescence is sudden and generally marked. The temperature may fall 1° or 2° below normal. Subnormal interval temperatures have occurred in a number of cases. Sweating may be profuse, scanty, or absent. It is commonly present. Vomiting often accompanies a paroxysm. A single paroxysm may occupy from six to eight hours. The intervals between the paroxysms may be fever-free or show a slight febrile movement.

The Onset—The onset is often like a simple malarial attack. The type may be established at once, or only after a period of irregular paroxysms. When first seen the patient may give a history of recent apparent malaria, with apparent relief from quinine. Such a history almost necessarily deceives one at first as to the true nature of the illness. It has probably led also to the false assumption of recent malarial attacks by some authors.

1. *Double Quotidian Type*—I have been able to find only one case in which this type was followed unchanged to the fatal termination. The double quotidian type has generally appeared only toward the end of the disease. The previous course may have been quotidian, tertian, or irregular.

Illustrative case—Coupland's Case I. Male, aged forty-one years. Gonorrhœa at twenty, otherwise healthy. Took cold two months ago. Next day had pain in left leg, which was relieved by local applications. Patient always felt weak afterward. Five weeks ago began to suffer from "shivering fits," from which he has not since been free. Paroxysms at irregular intervals, some every third day, some every fifteen hours. Thought he had ague. Each attack was accompanied by vomiting and was followed by an intolerable sense of heat. Became progressively weaker. No pain, but noticed latterly that legs swelled toward night.

Status Præsens—Looks very ill; is spare and sallow. Lungs practically normal. Cardiac dullness normal. Rough systolic bruit at apex. No thrill. Pulmonic second sound ringing; aortic dull.

Course—Throughout whole course, pyrexial periods averaged about five hours. Rigors, which sometimes commenced with and sometimes after the initial rise of temperature, averaged one and one-half hours' duration. The apyrexial periods, during which the temperature frequently fell to extreme subnormal levels, averaged ten hours. Absence of sweating was a marked feature.

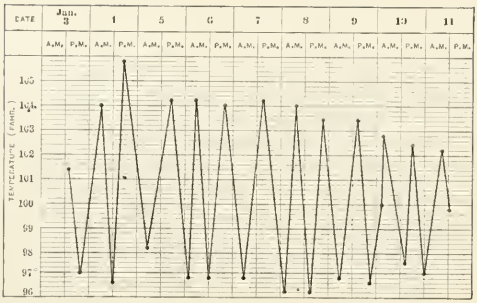

FIG. 1.
Double quotidian type. Coupland's Case I.

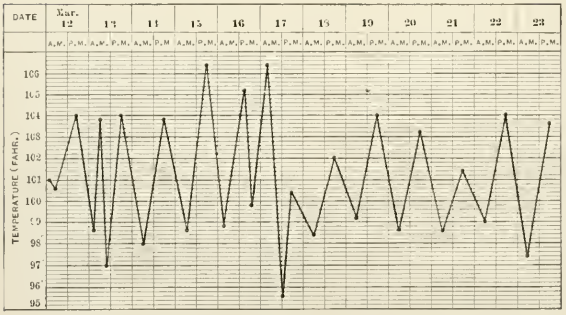

FIG. 2.
Double quotidian changing to quotidian. Coupland's Case II.

Fig. 1 represents the double quotidian paroxysms. Fig. 2 represents double quotidian changing into quotidian paroxysms.

2. *Quotidian Type—Illustrative Cases*—Dock's Case I. (1895). Female, aged twenty-six years. Forceps delivery, with laceration of perineum. Wound healed quickly. Three weeks later went sleighing. Afterward remained in bed. Had chills, fever, and sweating almost every day.

Status Præsens—Slight emaciation. Moderate effusion in right knee. Feet painful, but not swollen. Fugitive painful swell-

ings in fingers. Heart enlarged, impulse heaving, loud mitral systolic murmur. Second sounds normal. No cardiac lesion found at examination prior to administration of chloroform for forceps delivery.

Course—Rise of temperature usually occurred about noon, reached its acme about 5 P. M., sometimes one or two hours later, and after remaining at about the same point for one or two hours fell rapidly, usually reaching normal by midnight. Toward the last subnormal temperatures often occurred. Chills not constant, though very common, coming on early in the rise of temperature and of short duration. Sweating almost always present in the decline. Patient felt well, though weak, during apyrexial periods. Pain in the knees, ankles, feet, and hands, and itching of the skin were frequent. No cutaneous hemorrhages, but urine became bloody shortly before death. Fig. 3 shows the quotidian paroxysms.

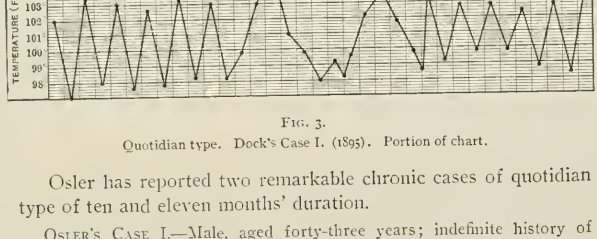

Fig. 3.

Quotidian type. Dock's Case I. (1895). Portion of chart.

Osler has reported two remarkable chronic cases of quotidian type of ten and eleven months' duration.

Osler's Case I.—Male, aged forty-three years; indefinite history of chronic malaria when a young man; illness began with a chill, followed by fever, malaise, and muscular soreness; headache, loss of appetite, insomnia, and cough were marked symptoms; spleen large; sympotms abated in a few days and patient resumed business; three weeks later marked dyspnœa, with increasing cough, developed, and a loud systolic murmur was heard at the apex for the first time; had daily intermittent fever, usually subnormal in the morning, and ranging from 102° to 103° in the evening, with occasional sweats; pains in different parts of the body, particularly in the left inguinal region; tenderness over the fourth and fifth left costal cartilages; fever continued throughout winter, with weakness, cough, and dyspnœa; patient in the Johns Hopkins Hospital from March 15 to May 10; there were no chills, only slightly chilly feelings; morning temperature was 97.5° to 98°, with a gradual rise to 102° to

103° at 4 P. M., and a gradual fall to normal at midnight; between 4 and 5 o'clock in the afternoon, sometimes not until evening, there was sweating, occasionally profuse; more frequently the skin was only slightly moist; the general conditions improved somewhat, and the patient gained slightly in weight. The only complaint was pain in the splenic region, sometimes with very distinct tenderness. There was no change in the cardiac condition while in the hospital. The temperature range was low, and often subnormal toward the end. Petechiæ appeared. Sweating continued. The patient failed progressively. Diarrhœa developed. The urine contained blood and blood casts. Death.

3. *Tertian Type—Illustrative Case*—Elsner's Case I. Symptoms of intestinal infection for several days. At the same time had a persistent gonorrhœal discharge. Without marked disturbances of any kind, without evidences of constitutional infection, patient suddenly developed a tertian form of intermittent fever, and ague was suspected. It became evident after the third day that the prostration was greater than in malaria, the delirium more marked, an nervous symptoms more profound. There was increasing shortness of breath. Heart rapid, sounds suggestive of mitral insufficiency. Spleen large. Albuminuria. After the third chill, in spite of the fact that the tertian type of fever persisted, the temperature was always slightly elevated. Physical signs of auriculoventricular insufficiency, with aortic stenosis. Death in fourth week, in typhoid condition, with hemiplegia, occasional chills, and sharp rises of temperature. Fig. 4 shows tertian paroxysms.

4. *Quartan Type—Illustrative Case*—Wenckelbach. Male, aged fifteen years. Entered eye clinic, after measles, for interstitial keratitis. Developed intermittent fever, complained of palpitation. Heart enlarged; sounds clear, but accentuated. Fever followed quartan type. Intervals not fever-free. No malarial parasites in blood. Daily attacks of palpitation, up to 160 per minute. Heart still further enlarged. Spleen enlarged, with piercing pains. Staphylococcus albus obtained from blood. Diagnosis, infective endocarditis. Cured after injections of collargol. Fig. 5 shows quartan paroxysms.

5. *Septan Type—Illustrative Case*—Henry. Female, aged nineteen years. Typhoid three years ago, rheumatism in childhood, pneumonia a few months ago. Pregnant. Well-compensated mitral obstruction. Repeated examinations of blood for malarial parasite gave negative result. Rise of temperature accompanied on two occasions by chill, once by vomiting. "The

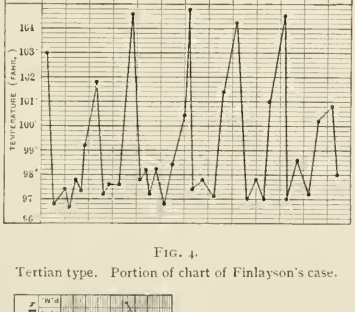

FIG. 4.
Tertian type. Portion of chart of Finlayson's case.

FIG. 5.
Quartan type. Wenckebach's case.

FIG. 6.
Septan type. Henry's case.

inference that the fever is dependent on the heart lesion is based on the fact that no other cause could be found for it." Patient recovered.

This case is the only one of pure septan type that I have been able to find, though septan paroxysms have occasionally occurred toward the end of cases which at other times had followed other types. Fig. 6 shows the septan type.

6. Mixed Types—These may be dismissed with a few words. The term is employed to designate only those cases which present two different types simultaneously. Such cases must be extremely rare. I have not been able to find even one which followed this course uninterruptedly. However, since Finlayson's case presented mixed tertian and quartan paroxysms during two months of its course, it seemed necessary to include the "mixed types." Fig. 7 is a part of the temperature chart of Finlayson's case.

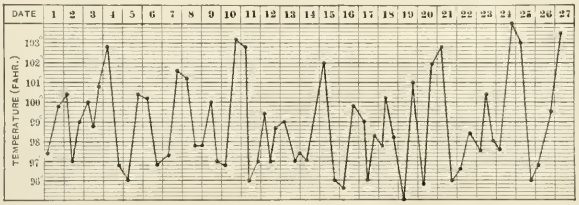

Fig. 7.
Mixed tertian and quartan types. Portion of chart of Finlayson's case.

7. Irregular Types—The irregular types constitute by far the majority of cases of infective endocarditis which bear resemblance to malarial fever. The resemblance rather dates back to the time, however, when exact knowledge of the malarial parasite had not been acquired—when cases were diagnosticated as malarial fever simply because of recurring periods of pyrexia. No less an authority than Dreschfeld states, in Allbutt's *System of Medicine*, that all cases of chronic infective endocarditis resemble malarial fever. Such a statement is, to say the least, unfortunate.

The irregular acute cases are generally so clearly septic and the constitutional symptoms so marked that there is little difficulty in recognizing them, while the chronic cases are generally so irregular and so frequently develop in the course of chronic

cardiac valvular disease that one is not left long in doubt as to their real nature.

Diagnosis—The diagnosis of a pseudomalarial form of infective endocarditis is often very difficult. As in other varieties, the heart may give neither signs nor symptoms. If the infective process has been engrafted upon an old valvular lesion, the diagnosis need not present much difficulty, nor if an atrium of infection can be found. But the cryptogenic cases, occurring in persons previously healthy, or in those who, living in suspicious localities, give histories of recent apparently malarial attacks, may severely tax the skill of the clinician. As already mentioned, the disease is subject to sudden and complete, though temporary, interruptions. The patient may have been taking quinine. Under the circumstances, what is more natural than to assume that the quinine has stopped the paroxysms, even though the blood may not have shown the malarial organisms? When the patient returns for treatment, or if he comes with such a history, the existence of malarial infection is presumptive, and the case may go unrecognized, at least for a time. Then the failure of quinine to mitigate or arrest the paroxysms gives the first intimation that the patient is not suffering from malaria—assuming, of course, that a complete examination of the blood has not been made. Such an examination will at once rule out malaria, but, on the other hand, the presence of the malarial parasite does not necessarily exclude infective endocarditis (*vide* Herzog's case).

In infective endocarditis there is generally though not always leukocytosis; in malaria, leukopenia is the rule, except during a paroxysm. In infective endocarditis the polynuclear leukocytes are increased; in malaria there is relative lymphocytosis. Pigmented leukocytes are found in nearly all severe and protracted or fatal cases of malaria. The red cells and hæmoglobin show much the same changes in both conditions—rapid, marked oligocythæmia, hæmoglobin reduction, and degenerative changes in the cells. The changes are essentially those of secondary anæmia, though sometimes in malaria they pass into the pernicious type. The recovery of the infective agent from the blood is often possible and always conclusive, provided no other nidus than the heart valves can be found.

One of the remarkable features of these forms of infective endocarditis is the strength of the patient between the paroxysms. Until the disease is advanced he may feel quite well, and the

classical picture of sepsis be lacking. Changes of type commonly occur during the course of the disease, and should be of much aid in diagnosis. In Ord's case, for example, the type changed from quotidian (two weeks) to tertian (two weeks), then to quartan, and later became irregular, the paroxysms recurring sometimes at intervals of a week. If an obstructive valvular lesion develops in a case pursuing a malarial course, the disease is almost certainly infective endocarditis. But, after all has been said, many cases will prove difficult to diagnosticate.

Pathological Anatomy—The simulation of malaria by infective endocarditis appears again in many cases in the pathological anatomy and histology of the viscera. In both diseases the long-continued hæmolysis results in the deposit of pigment in the spleen, liver, and bone-marrow, and while in malaria this pigmentation is much more extensive, owing to the activity of the parasites, nevertheless, the pigmentation of the viscera seen in septic processes often reaches a grade quite comparable to that of malaria. Thus, the spleen in septic processes may be considerably enlarged, distinctly brownish in color, and in very acute cases nearly diffluent. Likewise, the liver may exhibit a brownish or leaden color, much like that of malaria. Histologically, there is the same filling of endothelial cells of pulp and capillaries with masses of granular and globular pigment, the minute morphology of which is very similar in both malaria and infective endocarditis with bacteræmia. Hence it is unsafe to attempt to distinguish malaria from septic processes merely on the gross or microscopic appearance of pigment in the viscera. The only reliable method is the demonstration of the bodies of malarial parasites in cases of this disease, and their absence in septic processes. In nearly all other respects the pathological anatomy of pernicious malaria may be closely reproduced by septicæmia.

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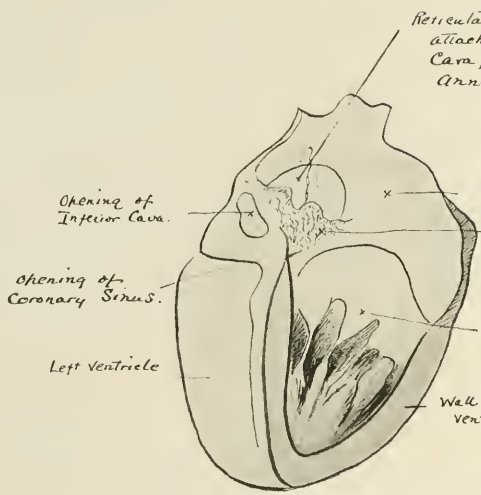
CONGENITAL MALFORMATION OF HEART—DEFECT IN INTERAURICULAR SEPTUM.

By A. M. Pappenheimer, M.D., *Pathologist.*
(From the Pathological Laboratory of Bellevue Hospital.)

The following case of an unusual congenital malformation of the heart in an infant of four months is of little clinical interest, inasmuch as the child was brought to Bellevue Hospital in a dying condition and was under observation only a few hours. On examination there was noted neither cyanosis nor clubbing; and no cardiac murmurs were heard. At autopsy, beside the cardiac lesion, there were found a maculo-papular rash over the entire body, rhagades about the mouth, and other evidences of congenital syphilis. The heart showed this interesting anomaly: The heart as a whole is distinctly enlarged, weighing 47 gms. There is very marked hypertrophy of the right ventricle (5 mm. on cross-section). The foramen ovale is very large, measuring 2 cm. in diameter, and is covered by a delicately reticulated curtain, which, when the heart is floated under water, balloons out into the cavity of the right auricle. The curtain has a crescentic, slightly thickened free margin; the cornua of the crescent are attached to the upper and anterior arc of the annulus, about 1 cm. apart, ending as slightly thickened cords upon the left auricular surface of the ring. The attachment of the curtain at its base follows the inferior margins of the annulus; delicate filaments also pass to the left margin of the entrance of the inferior cava. Along its lower attachment to the foramen ovale there is a trabeculated cord, which runs along the floor of the left auricle.

A second curtain, also built of a fine, lace-like, reticulated membrane, is attached at its base, by delicate filaments, to the right border of the inferior cava, and is continued forward a short distance along the inferior margin of the annulus, on its right auricular surface. The free border of this curtain, which is slightly thickened and more membranous in character, is therefore attached above to the vena cava at its upper portion, below to the inferior margin of the annulus. A thin, flattened filament courses from the upper portion of the border to become inserted upon the roof of the auricle, about ¾ cm. from the margin of the ring.


Reticulated curtain, partially closing annulus, attached to left border of opening of Int. Cava, and to left auricular margin of Annulus.
Right Auricle.
Opening of Inferior Cava.
Reticulated Curtain attached to right border of opening of Int. Cava, and to right auricular margin of annulus. A fibrous Strand runs from Edge of Curtain to roof of auricle.
Opening of Coronary Sinus.
Tricuspid Valve.
Left Ventricle
Wall of Right Ventricle

VIEW INTO CAVITY OF RIGHT AURICLE, SHOWING DOUBLE CURTAIN OVER ANNULUS.

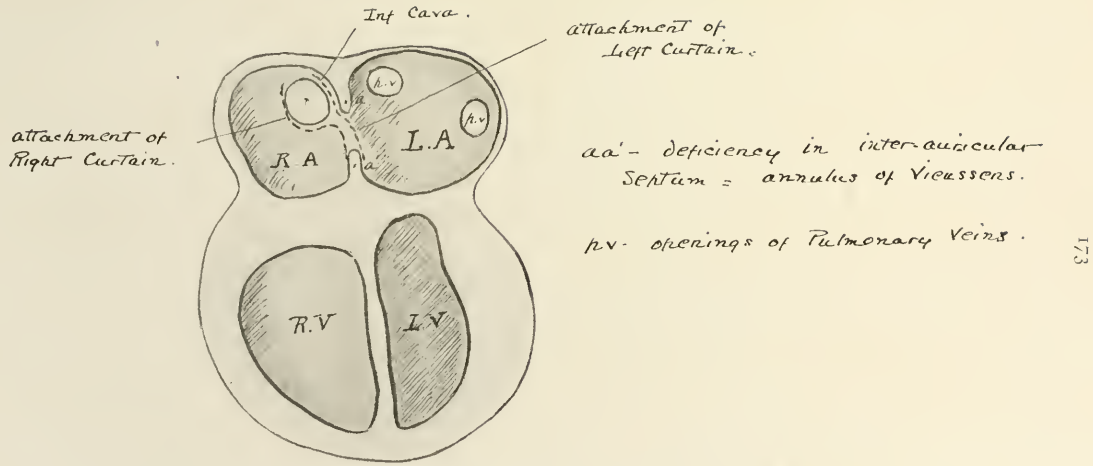


DIAGRAM SHOWING ATTACHMENT OF CURTAINS TO EITHER MARGIN OF THE OPENING OF THE INFERIOR VENA CAVA INTO RIGHT AURICLE.

The normal interauricular septum is therefore represented by two curtains attached at their base to the right and left borders of the inferior cava.

The tricuspid, pulmonary, aortic and mitral valves show no anomalies. The conus arteriosus is somewhat dilated and hypertrophied. There is no defect in the interventricular septum. The ductus arteriosus is closed. The aorta is of normal calibre throughout.

As to the frequency of this particular form of mal-development, I can give no figures. It is well-known that a slight patency of the foramen ovale at the upper and anterior margin is present in a large proportion of all cases—roughly, one-fifth; and larger defects in the foramen ovale are also amongst the most common of congenital defects. This specimen, however, shows an anomaly of a different type. The normal septum is represented merely by an annular ridge, and, instead of a single membrane, more or less covering the opening, we have two distinct reticulated curtains, overhanging the orifice; and attached to opposite margins of the opening of the inferior vena cava. In looking over a considerable number of reported cases of defects in the auricular septum, as well as the descriptions of cardiac anomalies, in the special text-books, I have not met with an analogous case.

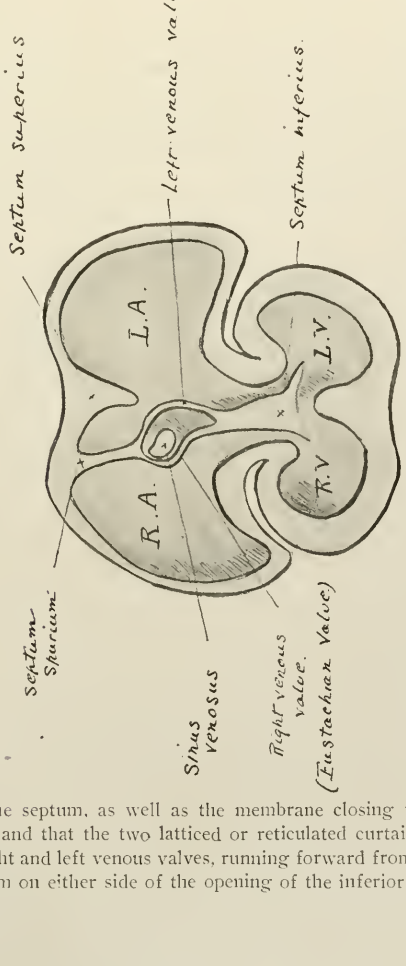
The chief interest in such a malformation lies in an attempt to explain it on embryological grounds. If I might venture such an explanation, I should say that the condition was produced in some such way as this:

In the human embryo of six or seven weeks, the opening of the sinus venosus into the common auricular cavity is guarded by two valve-like ridges, known as the right and left venous valves, which project into the cavity of the auricle. These pass above into a muscular fold, which is continued over the roof of the heart, and is called the septum spurium. This septum spurium later disappears, probably by fusing with the true septum atriorum. The right venous valve forms the crescentic fold of endocardium at the lower margin of the foramen ovale, known as the Eustachian valve. The left venous valve disappears.

The true septum grows downwards and forwards to fuse with the endocardial cushion from which the ventricular septum and auriculo-ventricular valves are formed. Before the completion of this interauricular septum, however, an orifice appears above and at the back of the septum—the foramen ovale. This becomes covered by a second membranous septum, the limbus of Vieussens,

which starts from the post-auricular wall a little to the right of the interauricular septum and grows forward over the foramen.

To explain the malformation present in this heart, we may suppose that there has been an agenesis or arrest of development of



HUMAN EMBRYO HEART (HIS) ABOUT SEVEN WEEKS, SHOWING RIGHT AND LEFT VENOUS VALVES, AND BEGINNING SEPTUM FORMATION.

the true septum, as well as the membrane closing the foramen ovale; and that the two latticed or reticulated curtains represent the right and left venous valves, running forward from the septum spurium on either side of the opening of the inferior cava.

TWO CASES OF STENOSIS OF THE AORTA AT THE ISTHMUS.*

By Alwin M. Pappenheimer, M.D., *Pathologist.*

(From the Pathological Laboratory of Bellevue Hospital.)

Case I.—For the clinical records of the first case I am indebted to Dr. Loomis. The lesion was an accidental finding at autopsy, and, as in so many of these cases, was unsuspected during life. As in addition to this stricture of the aorta, the patient presented some other rather interesting lesions, it may be worth while to give a brief abstract of his clinical history.

Family History—Negative; habits, excessive user of alcohol and tobacco.

Previous History—Usual diseases of childhood. Rheumatism, affecting joints of lower extremity, twelve years ago; malaria seven years ago.

Present Illness—Only a few facts could be obtained: About a month ago he entered the New York Skin and Cancer Hospital, because of increasing swelling of the abdomen, and the recent appearance of a purpuric rash all over his body. His temperature while at the Skin and Cancer Hospital ranged between 103° and 105°. A diagnosis of tuberculous peritonitis was made, and a laparotomy performed. Following this, his temperature diminished, but his general condition did not improve. On December 28 he was transferred to Bellevue, dying there four days later.

While at Bellevue Hospital his temperature was subnormal. Physical examination showed very marked emaciation. Over face, neck, trunk and arms, a blotchy purpuric eruption, the largest blotches not more than ½ cm. There was marked pulsation of the carotids. The left radial pulse was absent. The left hand was cold, discolored and purplish red. In the left cubital fossa in the course of the left brachial artery, there was a fusiform thickening of the vessel. Above this point pulsation; below, none. The right radial pulse was of poor force, large excursion, sudden impact. There was moderate thickening of the vessel. The femoral pulse of both sides was feeble and indistinct. No pulsation was obtained in the popliteal arteries, nor in the dorsalis pedis. No superficial pulsation was noted about the thorax or abdomen.

Heart—Apex beat in sixth space, 3¾ inches to the left of the median line. Action was irregular and not forcible. Left border was 4 inches from the median line; right border ¼ inch to right of sternum. The

* Proceedings N. Y. Pathological Society, New Series, Vol. V., No. 8, January, 1906.

Proceedings N. Y. Pathological Society, New Series, Vol. VI., No. 5, October, 1906.

sounds: A loud double murmur was heard at the base, less intensely over the rest of the chest, transmitted to the carotids. At the apex there was a short pre-systolic murmur, and a slight pre-systolic thrill.

Lungs—Showed signs of intense congestion at the bases.

Abdomen—Showed distention; signs of marked ascites.

Liver—Percussed to the fourth space above, and to free border below; edge not felt.

Spleen—Not palpable.

Blood Count—Red, 3,400,000; leucocytes, 57,000; haemoglobin, 50% differential: polymorphonuclear nutrophiles, 85%; lymphocytes, 7%; large amount of nuclears, 5%; transitional, 3%; no eosinophiles.

Urine Examination—Negative.

Patient's condition, which was almost moribund, became rapidly worse. Died four days after admission.

Autopsy—Performed fifteen hours after death, as follows: Small-framed male; marked emaciation. Purpuric rash over face, neck, trunk and arms, confluent over dorsum of left hand. Individual petechiae average about 3mm. Healed laparotomy wound, 1½ inches long, in median line below umbilicus. Slight oedema of feet. Marked rigor of lower extremities. Abdomen contains about three litres of clear, dark, amber fluid. Omentum adherent about operation wound. Intestinal serosa shows numerous small, haemorrhagic blotches. A few also on parietal and diaphragmatic perintoneum. Parietal peritoneum is thickened and opaque. Liver reaches a hand's breadth below costal margin. Omental fat atrophic, and shows post-mortem discoloration. Diaphragmatic vault at fifth right rib; sixth left rib. Appendix retro-coecal. Thorax: Anterior mediastinal glands behind ensiform enlarged; show few miliary tubercles on section. Left lung adherent to parietes posteriorly. Right lung shows complete synechia. Pericardial sac contains normal amount of clear fluid.

Heart—Visceral pericardium spotted with punctate petechiae. Epicardial fat is scant. Right heart contains fluid blood. Tricuspid ring admits three fingers. Valves are normal. Foramen ovale closed. Pulmonary valves are normal. Myocardium of right ventricle shows slight hypertrophy. Mitral ring admits three fingers. Left ventricle shows moderate dilatation and flattening of papillary muscles. Mitral segments show slight diffuse thickening. The chordae are shortened and thickened. Aortic valves show an anatomical anomaly, there being but two large cusps, one anterior and one posterior. These valves are thickened and fused at their junction, for a distance of ¾ cm. At their left junction there is marked nodular thickening, possibly representing the corpus arantii of the missing valve. There are a few minute pin-point vegetations along the edge of each cusp. Extending downwards on the ventricular wall, for a distance of 5 cm., the endocardium shows extensive erosion of the intima. Eroded areas are the seat of large verrucous unorganized thrombotic deposits, which in their central portion have undergone puriform softening. The myocardium of the left ventricle is hypertrophied, averaging 2½ cm.; firm in consistency. Brownish in

color. The coronaries are tortuous. Intima shows plaques of atheromatous thickening. Lumen, however, patent. The ascending and transverse aortae are normal, save for a few small atheromatous plaques. Immediately below the origin of the left subclavian, the intima is thrown up into a ridge, which partially encircles the aorta. There is at this point a moderate constriction, the circumference measuring 3¼ cm. A second shallow ridge runs downward, joining this on the posterior wall; 2 cm. below this point there is a very marked annular constriction of the lumen, the aorta being thrown up into a fibrous ridge or diaphragm. The circumference at this stenosis is about 1½ cm., and in the unopened condition, the diameter about 4 mm. Between these two constrictions, and about ¾ cm. above the lower, there is a shallow evagination or dimple in the aortic wall, representing the original point of entrance of the ductus arteriosus. A similar depression is seen in the wall of the pulmonary artery in a corresponding situation. Between the two there can be traced a well-defined strand of fibrous tissue, representing the remains of the ductus arteriosus, which is completely closed. Below the lower constriction, the aorta rapidly resumes its normal calibre, measuring 6 cm. in circumference in the upper thoracic portion—5½ at its junction with the abdominal. A collateral circulation has apparently been established by way of the internal mammaries, and deep epigastric arteries. The internal mammaries are greatly dilated, their circumference about 2½ cm. The deep epigastric arteries also are obviously enlarged. Details of collateral circulation via the branches of the sub-clavian, scapular, long-thoracic and intercostal arteries, not worked out.

Lungs—Left lung: Pleura has numerous petechiae. Bronchi congested. Contain frothy mucus. Lymph nodes enlarged; anthracosed. Old tuberculous scars at the apex. On section upper lobe shows encapsulated cheesy nodules, size of hazel nut. Upper portion lower lobe shows a diffuse lobular pneumonia, with diffuse miliary abscesses in upper portion. Large amount of exudate can be scraped from surface.

Right lung: Pleura greatly thickened over lower lobe, especially on the diaphragmatic surfaces. On section, there is general oedema of all the lobes. Marked interstitial fibrosis.

Liver—Weighs 4 lbs. Capsule is thickened, and opaque. Surface granular, with marked perilobular injection, which is irregularly distributed. The tissue is of increased firmness. Parenchyma is pale.

Spleen—Enlarged to three times normal volume, approximately. Weighs 1 lb. 12 oz. On the surface are seen about ½ dozen raised, roughly circular areas, the largest situated near the pole, 2½ cm. in diameter. These show a yellow centre, with a pink periphery, and on section, correspond to healed anaemic infarcts surrounded by a zone of hyperaemia. The splenic tissue is pale, firm and glazed. Neither trabeculae nor follicles are prominent.

Pancreas—Large, firm and cirrhotic. Splenic artery shows atheroma and thickening.

Adrenals—Congested.

Kidneys—Left kidney small and plac. Capsule splits on stripping. Surface shows numerous punctate haemorrhagic spots. A few small cysts. Cortex diminished in width. Color pale. Vessels irregular. Markings indistinct. Pelves normal.

Right kindney shows similar lesions.

Stomach—Shows hour-glass constriction. There is a small superficial erosion of the mucosa at the junction of the cardia and the oesophagus, about 1½ cm. in diameter. The epithelium of the oesophagus is macerated and desquamated.

Small and Large Intestines—Normal.

Bladder—Shows pin-point sub-mucous haemorrhages.

Mesenteric lymph nodes are enlarged, pale and firm. Left brachial artery in the cubital forsa contains an embolism which in places is slightly adherent to the endothelium.

Head—Not opened. Permission refused.

Anatomical Diagnosis—Endocarditis, acute infective verrucous; valvular disease, chronic cardiac (aortic insufficiency); anomaly of aortic valves; stenosis of isthmus of aorta; pneumonia, lobular; pleurisy, chronic adhesive; embolism left brachial artery; cirrhosis of liver; hydroperitoneum; infarcts of spleen.

Dr. Carlisle has kindly allowed us to make use of the clinical records in this case.

Case II.—F. B., machinist; age 64 years; heavy wine drinker; usual diseases of childhood.

Present Illness—Four months ago swelling of abdomen; previously, occasional morning vomiting; one week ago, swelling of feet; beginning with feet, gradually involving entire legs and genitalia; at this time also, rapidly increasing dyspnoea.

Physical Examination—Upper extremity and face emaciated; marked oedema of lower extremities and genitalia; distension of abdomen and ascites.

Radial Pulse—Normal in rate, regular, moderate volume and tension; considerable sclerosis of arteries.

Heart—Apex beat not seen or felt. Sounds heard with maximum intensity in fifth space, 4 inches from median line. First sound valvular in quality. No apical murmurs. Basic sounds very indistinct. Over second left interspace a harsh systolic murmur, not transmitted, but "heard well behind, between scapulae on left side."

Lungs—Signs of fluid over both bases, and emphysema.

Liver—Dullness diminished; spleen not palpable.

Urine—Specific gravity 1019; no albumen or casts; trace of bile.

Blood Examination—Negative.

Delirium developed; oedema rapidly increased; death after four days, with oedema of lungs.

The necropsy was performed two days after death by Dr. Flournoy. In addition to an advanced atrophic cirrhosis of the liver, with ascites and general anasarca, there was found this interesting vascular lesion.

Heart—Slightly enlarged, apex being formed by left ventricle. Right heart, with its valves, is normal. Wall shows no hypertrophy. Foramen ovale is closed. The pulmonary artery, above the sinuses measures

CASE I.

Stenosis of Aorta at Isthmus.

aa'—Upper constriction.
bb'—Lower constriction.
L—Ligamentum Botalli.
D—Entrance of Ductus Arteriosus into Aorta.
P—Pulmonary Artery.
I—Innominate Artery.
Lc—Left Common Carotid.
S—Left Subclavian.
T—Thoracic Aorta.

75 mm. Wall is thin, measuring 2 mm. Wall of the left ventricle is hypertrophied, measuring from 2 to 2½ cm. Mitral valve is normal, save for very slight thickening of the edges. The aortic cusps are normal. Left coronary artery is enlarged and tortuous, standing out prominently, and at a point about 3 mm. from its commencement it is almost completely calcareous. The descending branch is completely closed. Right coronary artery is likewise calcareous, thickened and dilated. Aorta measures, just above the sinuses, 85 mm. in circumference. Wall 2½ mm. in thickness. The ascending portion of the arch appears slightly dilated. The transverse portion is of normal width. Just beyond the origin of the left subclavian artery there is a tight stenosis, which barely admits the passage of a small pencil. This appears on the outside as a more or less gradual constriction, but on opening the aorta there is found a thick ridge of tissue, completely encircling the aorta, like a diaphragm, about 3 mm. distal to the lower margin of the orifice of the left sub-clavian artery. The circumference of the aorta at this point measures on its internal surface about 20 mm. The intima of the ascending portion of the aorta is smooth, save for a few small plaques of fatty degeneration, just above the sinuses. On the proximal side of the stenosis, however, the intima is the seat of extensive calcareous deposits, most marked on the inferior surface, opposite the orifices of the great vessels. Immediately below the stenosis the aorta very rapidly assumes its normal calibre, and at a distance of 3 cm. distal to it measures 8 cm. in circumference. The wall of the abdominal aorta is thin, and there appears to be little or no degeneration of the intima.

The depression which corresponds to the entrance of the ductus arteriosus into the aorta is slightly marked, and is situated about 3 mm. below or distal to the constriction. The ductus itself is obliterated throughout, but can be seen as a distinct fibrous cord joining the wall of the aorta at a very acute angle.

The internal mammary arteries were on both sides greatly enlarged, somewhat tortuous and calcareous, and measured 15 mm. in circumference. The great vessels of the neck all appear much dilated, the right subclavian, for example, measuring 29 mm. in circumference. The superior intercostals are especially enlarged, the one on the left side measuring 19 mm. in circumference.

As in the majority of these cases of aortic stenosis, the condition proved to be entirely an accidental finding at autopsy, and was of no clinical significance whatever. The collateral circulation appeared to be entirely competent.

Stenosis at the isthmus of aorta is not a great pathological rarity. A series of cases has been collected by Marié, Vierordt and others. Bonnet, in an exhaustive article in the *Rev. de Med.*, 1903, has abstracted all the previous cases, and has discussed very thoroughly, the pathology and possible causation of the condition. In all, there have been about 160 cases recorded, with autopsies. Of these, 105 were in adults, and 55 in new-born children.

Clinically, it is doubtful whether this condition ever causes marked symptoms. In those cases where the condition has been uncomplicated by other cardiac lesions, there has been no circulatory disturbance in the lower extremities, and no cardiac hypertrophy. However produced, the obstruction takes place so slowly as to afford ample time for the establishment of a competent collateral circulation. In the absence of subjective symptoms, caused by the stenosis, the diagnosis has nevertheless been made in several instances during life. Excluding aneurysm and embolism of the aorta, it would seem that an absence or marked diminution in the size of the femoral pulses, as contrasted with the carotids and radials, and the presence of pulsating superficial arterial branches in the thoracic region, suggesting a collateral arterial circulation, would point to this condition.

The stenosis regularly occurs at some portion in the isthmus of the aorta, or that portion between the fourth and fifth branchial arches. The stenosis is of two general types: Either a narrowing, involving the entire isthmus, and therefore of considerable extent, or a localized constriction of the lumen, situated in the region of the entrance of the ductus arteriosus. The former type is seen, especially in the new born, and is considered by Bonnet as a simple *persistence* of the narrowing, which is a constant anatomical feature of the aorta at this point at birth, the so-called Isthmus of His; or, where it occurs in somewhat later life, as a simple *arrest of development*. Occasionally, where the stenosis above the ductus arteriosus is an extreme one, still more rarely, where there is a complete atresia above the ductus, he assumes that the condition dates back to a very early period in foetal life. The other type, that is, a stenosis localized at or near the entrance of the ductus arteriosus, is the one most commonly met with in adults. Here we have a ridge of new tissue projecting into the lumen of the aorta, forming a diaphragm. The condition has been compared to the effect produced by the constriction of a ligature at this point.

To explain the causation of the stenosis, various theories have been advanced. One of the oldest was that of Hamernik. He assumed that the causation varied with the relation of the ductus arteriosus to the stricture. Where the stenosis occurs above the entrance of the ductus Botali, it is due to a simple persistence, or arrested development of the isthmus. Where the stenosis was at the point of insertion of the ductus, we have a true anomaly or

congenital stricture. The occurrence of a stenosis below the entrance of the ductus he explained by the organization of the thrombus, which is formed during the obliteration of the duct being continued into the aorta, and in its cicatrization producing a constriction. This last theory has been abandoned, since cases of stenosis occur at this point, in which the ductus remains patent.

Rokitansky assumes a congenital slenderness or narrowing of the aorta, at the level of the isthmus; the aortic wall being more tenuous than normal, is less resistent to the traction of the ductus arteriosus. In consequence, a stenosis develops.

Skoda first divided the cases of stenosis into two main groups; one where the stricture dates back to foetal life, and the second, described as the adult type, in which the influence of the ductus arteriosus is the predominating factor. He points out that the tissue of the ductus arteriosus is continued into the wall of the aorta, and for this reason, its contraction may act as a ligature. The variations in the relation of the ductus to the site of the stricture is explained by the variations in the angle at which the ductus enters the aorta, and the excess of fibrous tissue above or below the point of entrance, as the case may be.

1. Fetal type. 2. Fetal type. (Complete atresia.) 3. Adult type. (Usual form) 4. Adult. (Specimen.)

The first specimen does not fall readily into either of the two general types described. There is a linear stenosis both above and below the insertion of the ductus, and a moderate narrowing of the intervening portion. The lower ridge corresponds well enough to the usual type, as seen in adults. The significance of the ridge, which is situated about 1½ cm. above the entrance of the ductus, and immediately below the origin of the sub-clavian, is not clear. Whether we are to consider it as marking the beginning of the isthmus and the narrowing between it and the lower ridge, as a reversion to the foetal condition is not clear. Possibly it may be in some way related to the presence of the branchial arch described by Zimmerman, and other embryologists, as oc-

curring during an early period of foetal development between the fourth or aortic arch, and the fifth or pulmonary arch. It seems certain that the upper stenosis cannot in any way be explained by traction exerted on the part of the ductus arteriosus.

The second case conforms to the usual type found in adults, namely, a linear stenosis, situated a few millimeters above the entrance of the ductus arteriosus.

Although Bonnet states that cardiac complications are not more frequent than normal, in these cases, a hasty survey of the cases collected by him would lead me to the belief that this is not the case. An unsual number of his cases showed valvular changes, or acute endocarditis, especially at the aortic orifice. Moreover, in six (6) of his cases, the same anomaly of the aortic valves, namely, absence of one cusp, was noted. In those cases observed in the new born, one usually finds associated, very grave congenital abnormalities of the heart and arteries.

THE BACTERIOLOGY OF GONOCOCCUS INFECTIONS.*

By Thomas Flournoy, M. D., *Pathologist.*

(*From the Pathological Laboratory of Bellevue Hospital.*)

In compliance with the limitation implied in the title of this paper, my remarks shall be confined in general to the purely bacteriological aspect of infections with the gonococcus, and in particular to those points which are of greatest interest to the clinician. I shall not attempt to describe in detail any of the types of infection due to the presence of this micro-organism, but rather to summarize as briefly as possible our present knowledge of the characteristics by which the gonococcus is known.

The work of Neisser and of those who immediately followed him is accepted as having demonstrated the direct etiological relationship of this micro-organism with the condition clinically known as gonorrhœa. Further investigation indicated that attendant clinical phenomena, such as disturbances arising in the articulations and tendon sheaths and in the circulatory system, as well as elsewhere, are due to the presence of the same micro-organism. This latter statement has been questioned more than once, the opposite view attributing the symptoms in at least some of this class of cases not to the presence of the micro-organism itself, but of its toxins. This discussion, however, shall not concern us at present. We need only consider the statements of many observers who report the finding of gonococci in these comparatively unusual situations.

Micrococcus gonorrhœae is mostly simply described as a flattened diplococcus which decolorizes according to Gram's method. While this description practically sums up the morphology and staining reaction, it is unfortunately not sufficient to differentiate the gonococcus from all other bacteria. These two characteristics are the same in the species Micrococcus meningitidis of Weichselbaum and Micrococcus catarrhalis of Pfeiffer. It is necessary, therefore, to determine certain other characteristics before the identification of the gonococcus can be said to be placed beyond question. These further characteristics

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relate to its behavior when transplanted to artificial culture media; and, following the precept of those who first worked in its cultivation, the diagnosis of gonococcus rests chiefly upon one point—its inability to remain alive upon media which do not contain, in unchanged state, certain body fluids. These media are briefly designated as serum media and contain uncoagulated ascitic fluid, hydrocele fluid, or the like.

The complete definition of gonococcus, then, as ordinarily accepted is that of a flattened or biscuit-shaped diplococcus which does not retain the gentian violet of Gram's stain and does not grow outside the body except when supplied with certain media. Owing to this last requisite, the diagnosis of gonococcus by means of stained films from the infectious material cannot be regarded as conclusive. Assuming the above statement to be correct, and disregarding for the moment the practical importance of stained films in the routine diagnosis of gonorrhœal urethritis, it may be stated absolutely that, for the definite diagnosis of the presence of gonococcus, cultural methods must be employed. The presence of groups of micro-organisms within the pus cells from any infected part is not characteristic of gonococcus alone. The statement is made in all text-books upon bacteriology, that, for medico-legal purposes and for exact bacteriological determinations, cultivation must be resorted to.

In the clinical diagnosis of acute gonorrhea without complication, the necessity of any such complicated procedure does not exist. The diagnosis of gonococcus infections in situations remote from the uro-genital tract, however, is much more difficult than is generally supposed. The so-called meningococcus has repeatedly been isolated in blood cultures and Micrococcus catarrhalis is reported as having been isolated from the heart blood and from all the organs at autopsy in at least one case The meningococcus has been reported as occurring in the knee-joint, and other unidentified Gram-decolorizing diplococci have been found in the same situation. More important still, however, is the fact that in cases of joint affections complicating urethritis and clinically accepted as gonorrhœal in origin, there have been obtained from the affected joints diplococci which cannot be regarded as gonococcus if we accept the classical requirements for the identification of that species. Some of these micro-organisms cannot be distinguished from Micrococcus meningitidis, while others are merely reported as differing from

gonococcus in their ability to grow upon plain media without the addition of serum.

Thus far we have accepted without question the statement that gonococcus does not grow except upon the special "serum" media. It stands, however, by no means above question and many investigators have reported their successful attempts at cultivation of the gonococcus on plain media. While the merits of these individual reports are difficult to judge, they cannot be ignored solely on the plea that such successful attempts preclude the possibility of these strains, being true gonococci. Since the identification rests upon this negative qualification, it is subject to possibility of disproof at any moment. From time to time various European observers have recorded experiments in the course of which they have succeeded in growing gonococcus on plain media. Quite recently a French observer has reported a series of successful attempts at growing gonococci on plain agar.

Unfortunately, attempts at differentiation by means of specific agglutinative sera have failed to yield uniformly satisfactory results with this morphological group, so that this possible means of differential diagnosis is not available at present.

During the past year, under the direction of Dr. Norris at Bellevue Hospital, a considerable number of cases of the various types referred to have been studied bacteriologically, and the results of the various attempts at cultivation may be said to be of very considerable interest in the light of the classical dictum regarding the gonococcus.

The first class of cases to attract attention was a series of affections of the joints following urethritis. Of these cases it may be said briefly that, while morphologically identical with gonococcus, all of the species of micro-organisms isolated grew upon so-called plain media and were accepted as unclassified types, with the exception of one strain which agreed with all tests for meningococcus.

In order to secure further data for the comparison of these species, cultures were made from cases of acute gonorrhœa in the wards and in the out-patient department of the hospital, and a fairly large number of strains of Gram-decolorizing diplococci were obtained. It may be stated roughly that about fifty per cent. of these strains grew well on glycerine-agar in early and in subsequent generations. Circumstances prevented the persistent carrying out of attempts to cultivate the refractory strains on this

same medium, so it may only be said that repeated attempts were unsuccessful with certain cultures. Others, however, grew for weeks on glycerine-agar alone, beginning with the second or third generation outside the body.

The question of pathogenesis could not be answered for these strains, but in order to increase the chance of securing possibly true gonococci from any one case, not one but from three to six colonies were often fished from the original cultures and carried on as if they had been separate strains. No variation was noted, however, between the individuals from any one case. Each of these cultures resembled those of gonococcus in all the minor details of its cultivation. The appearance and consistence of the colonies on serum-agar were like that ordinarily described as typical for gonococcus, and the slight variations in morphology noted in old cultures of gonococcus were also present in these cultures after the first twenty-four hours. They were, then, like gonococcus in all respects and still were able to grow on plain media, while all of them were derived from cases of true clinical gonorrhœa.

If from a single comparatively small series of cases general conclusions may safely be drawn, two possibilities exist on the basis of the above facts. Either there may be present in urethritis beside the gonococcus, another more easily cultivated similar diplococcus, or the gonococcus, contrary to the accepted belief, is capable of growth on other than the serum media. What is true of urethritis is true of its complications, since it is hardly tenable that the same micro-organism can vary its biological characteristics with its change of locality in the body.

The clinician may safely make the diagnosis of gonorrhœa in acute cases upon the evidence of Gram-stained smears alone. The bacteriologist cannot accurately identify the gonococcus in the light of our present knowledge of its characteristics. He can, however, with certainty identify the meningococcus, whatever its source, and he can differentiate further certain other similar diplococci which are classed as Micrococcus catarrhalis.

ISOLATION OF GRAM NEGATIVE DIPLOCOCCI IN THREE CASES OF ARTHRITIS, ACCOMPANYING URETHRITIS; IN A FOURTH CASE, WITHOUT URETHRITIS.*

By Thomas Flournoy, M. D., *Pathologist.*

(*From the Pathological Laboratory of Bellevue Hospital.*)

The following cases have been observed in Bellevue Hospital during the past year, and the results of the bacteriological examinations have been considered unusual enough to warrant their being reported.

Dr. S. Alexander, Dr. C. H. Chetwood and Dr. H. M. Biggs have kindly placed the clinical data at our disposal.

Case I.—A. M., printer; 23 years of age; was admitted to the hospital in the service of Dr. Alexander, December 22, 1905, three weeks after the onset of urethritis, with purulent discharge, ardor urinae and frequency of micturition. On the day before admission, patient noticed pain in right knee, followed, during the night, by swelling of the joint. No history of trauma was obtained.

At the time of admission, patient complained of pain upon sudden movement of the knee, and of tenderness on both sides at the line of articulation. Examination showed restriction of voluntary movement, normal range of passive motion, and no swelling of the joint. Overlying skin normal. Moderate amount of yellowish discharge from urethra. Patient appeared to improve slightly under treatment; but on the fourth day after admission temperature rose, and knee became more painful, and during the succeeding ten days became red and swollen, with much effusion in and about the joint.

Thirty-four days after the first symptoms of arthritis, a small amount of turbid yellow fluid was withdrawn by aspiration, and sent to the laboratory. At this time, urethral discharge had practically disappeared.

Microscopic examination of the joint fluid showed numerous pus cells and a few groups of intracellular Gram-decolorizing diplococci; also numerous faintly staining Gram-negative bacilli. Ascitic agar plates streaked with floccules from the fluid showed, after 24 hours, eight or ten colonies of diplococci, decolorizing by Gram method of staining. Besides these, the plates contained a few micro-organisms of varying morphology and staining reaction, which, with one exception, were

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believed to be merely contaminations. Only one colony was found which was made up of bacilli resembling those found in stained preparations from the joint fluid, and this growth was unfortunately not further investigated.

The colonies of diplococci were small, grayish, viscid and translucent, and under the lens gave the appearance which is considered typical of colonies of gonococcus; that is, they were gray, with a slightly brownish tint, had fairly even circular border, and a moderately dense, coarsely granular, central portion, with lighter, finely granular peripheral zone. In the third and subsequent generation, transplants were made to glycerine agar, and to ascitic-agar, in the following manner: The entire colony was picked up on a broad platinum streaker and spread over the surface of the glycerine-agar plate, and then, without reinoculating, over the ascitic agar. On the glycerine-agar, a moderate, rather retarded growth was obtained in the fourth generation; and from these plates three further generations were obtained, showing somewhat more abundant growth on the same medium. In view of the number of generations and the increased amount of growth with subsequent transplantations, we can exclude the possibility of our having transferred from the serum-medium, with the micro-organisms, enough of that medium to insure continuation of growth.

On January 18, forty-seven days after the onset of the arthritis, a second aspiration was performed, and 15 cc. of turbid yellow fluid withdrawn. Stained films from this specimen showed pus-cells and intracellular Gram-decolorizing diplococci, without other bacteria. Cultures on ascitic-agar yielded, in this instance, about thirty colonies of Gram-decolorizing diplococci, without other micro-organisms. From the original plates (first generation) seven colonies were streaked upon glycerine-agar and then upon ascitic-agar plates. All of these transplants yielded a fair growth and from the glycerine-agar plates third and fourth generations were easily obtained upon the same medium.

The cultures from the two separate aspirations from this case were then transplanted upon Loeffler's blood-serum, gelatin and broth. Blood-serum tubes showed, after 24 hours, good growth in the form of small, raised, moist, grayish-white colonies, and inoculations from these to the same medium yielded always good growth. Broth tubes showed, after two to three days, uniform cloudiness with coarse, somewhat granular deposit. Transplants, made after four or five days, showed the organisms to be still living. No growth was obtained in gelatin.

Case II.—A. T., machinist; 27 years of age; admitted to Dr. Chetwood's wards on January 1, 1906; had contracted a urethritis two weeks before admission. Gave history of a previous attack of rheumatism, lasting for one week, part affected not stated. Denied previous attacks of venereal disease of any kind.

Three days before entering hospital patient injured his left knee in a fall, but continued working during the next day and a half, the joint meantime being painful, and beginning to swell. Two days after the injury pain became severe and swelling interfered with movement of the leg. Physical examination showed swelling, redness and calor of left

knee joint, with restriction of both active and passive movement. Inguinal lymph nodes enlarged and hard. Temperature 102°.

Fourteen days after onset of arthritic symptoms, a small amount of flocculent fluid was withdrawn from the joint by aspiration.

Microscopic examination showed presence of pus cells and of numerous intra and extra-cellular Gram-decolorizing diplococci. Cultures upon ascitic-agar plates gave pure growth of diplococci, similar to those found in stained preparations. The colonies on these plates were in all respects like those described and depicted for gonococcus.

Following the method previously described, transplants were made to glycerine and to ascitic agar. Although each generation after the first was tried in this way, no growth was obtained on glycerine-agar during the first seven generations. Inoculations upon Loeffler's blood-serum, however, gave good growth, when first tried (eighth generation), and sub-cultures from these tubes gave good growth upon the same medium. Transplantation to ordinary nutrient bouillon resulted in slight clouding of the medium and moderate flocculent deposit, but further bouillon tubes inoculated with these remained sterile. No growth was obtained in gelatin.

Case III.—L. G., colored woman; 17 years of age; married; admitted February 19, 1906, to Dr. Chetwood's service, three days after bearing a healthy male child. About ten days before coming to hospital patient had what she described as a boil on one of the labia, unaccompanied, she said, by vaginal discharge. About three days later her right knee became swollen, hot and painful.

Physical examination shows glands of Bartholin to be swollen and apparently tender, with small floccules of pus at the openings of the ducts.

Inguinal lymph nodes large and tender.

Nine days after onset of symptoms of joint involvement 12 cc. of turbid yellow fluid were withdrawn by aspiration. Smears showed presence of large numbers of pus cells, but no micro-organisms were found. Streak plates of ascitic agar, however, yielded pure culture of a Gram decolorizing diplococcus, morphologically like gonococcus. Cultures from floccules of pus in vaginal discharge yielded considerable number of colonies of Gram-decolorizing diplococci.

Most unfortunately, all cultures from this case were lost before observations of their behavior upon other than serum medias could be made, and their identity remains, therefore, unknown.

Case IV.—H. P., male; age 31; admitted April 18, 1905, to the wards of the Third Medical Division, service of Dr. Hermann M. Biggs.

This patient denied having had venereal disease, though he admitted great sexual and alcholic excesses during the week previous to onset of illness. Symptoms began April 10, with malaise, dizziness, weakness and gradually increasing pains in both legs, these pains finally becoming most marked in the right knee.

Physical Examination—Showed well developed and well nourished male; skin hot and dry; marked general pallor; mucous membranes pale. Visible pulsation in vessels of neck. Lymph nodes palpable.

Skin over right leg smooth and shiny; marked tenderness along popliteal nerve, and at the head of the tibia. Right leg cannot be voluntarily extended.

Heart sounds of fair force. Faint blowing systolic murmur at apex; not transmitted. Faint systolic murmur at base, heard loudest in vessels of neck. Pulse rapid, regular, fair force and volume.

Area of splenic dullness large. Organ palpable on deep inspiration.

Five days after admission the following note was made: Right knee swollen, hot, tender and painful. Evidence of fluid in the joint. Right ankle also tender and painful. Hip swollen and tender. Cardiac murmurs are more distinct. Temperature 102°. Pulse 132. Respiration 28. Fifteen days after onset of joint symptoms blood cultures were made, and knee joint fluid withdrawn for examination.

At this time, examination of the urethral secretion, as well as of the urine, by both microscopic and cultural methods, failed to give any evidence of gonorrhoea.

Microscopic examination of the turbid joint effusion showed large numbers of pus cells and a few groups of flattened, Gram-decolorizing diplococci within the pus cells. Ascitic-agar streak plates inoculated with the fluid gave pure culture of diplococci similar in morphology to those seen in the smears, decolorizing with Gram's method of staining.

In our hands, attempts to grow these diplococci upon other than serum media were unsuccessful during ten or more generations. Transplants given to Dr. Elser, however, have finally yielded a growth in ordinary nutrient bouillon.

The plates made from the patient's blood, a mixture of blood and agar in a proportion of about one to five (1:5), showed, after 48 hours, a few minute deep colonies, examination of which showed them to be made up of diplococci morphologically identical with and staining like those isolated from the joint fluid. Sub-cultures from these, were, however, unsuccessful, so that no identification could be made.

General considerations based upon the study of the micrococci isolated from these few cases bring out some interesting facts. In the first place, because of their cultural characteristics, none of these strains can be held to be gonococci, since they have all been grown upon culture media believed to be unsuitable for the growth of the true gonococcus.* Moreover, these cultures vary among themselves in a manner to be described below.

We have, therefore, micro-organisms from cases which are clinically gonorrhœal arthritis, and yet these micro-organisms are not gonococci, and moreover, are not of the same species. The examination of stained preparations made directly from the fluid aspirated in any of these cases would perhaps ordinarily have been taken to be sufficient to establish a diagnosis of joint infec-

* The micro-organism isolated from the third case is of course disregarded here, since study of its cultural characteristics was not carried out.

tion with gonococcus. Cultural methods, however, show that the diplococci seen in smears are not gonococci.

From each of these cases, then, there were isolated from the affected joints diplococci which, in morphology and staining reactions, bore a close resemblance to the gonococcus. Besides their ability to grow upon Loeffler's blood serum or in nutrient boillon their behavior upon certain other media merits special remark.

The diagnosis of the gonococcus appears to rest essentially upon the exclusion of the other similar diplococci. Following the researches of Neisser, Steinschneider and others, it has come to be generally accepted that the gonococcus, besides being always decolorized after Gram's stain, is capable of growth only on media containing proteids, *i. e.*, blood serum, ascitic fluid, hydrocele fluid, etc. The differential diagnosis between Micrococcus catarrhalis and meningococcus, from sources other than the spinal canal of patients suffering from meningitis is still somewhat uncertain. The chief points of difference so far brought forward are slight, being purely cultural and dependent upon appearance of growth and of colonies; and these characteristics are moreover admittedly variable and cannot fail to be modified to a greater or less extent by fluctuation of conditions pertaining to the medium employed. Further, it is extremely probable that the specific name Micrococcus catarrhalis has heretofore served to cover not one species, but all the members of an uncertain group of Gram-decolorizing diplococci which are neither gonococci nor meningococci.

Attempts at differentiation by means of specific agglutinative sera have yielded uniformly unsatisfactory results in the hands of others as well as ourselves, and up to the present time there has been reported no procedure which renders agglutination tests applicable for this class of micro-organisms. For these reasons, any means of ascertaining the species to which any particular culture belongs has a definite value.

During the past twelve months all cultures of Gram-negative diplococci obtained by us from whatever source have, at Dr. Norris' suggestion, been inoculated into the various carbohydrate-serum-water media, in the hope of obtaining from a classification of their reactions some means of identifying the various members of this morphological group.

The reactions obtained have been extremely uniform for each species except the so-called catarrhalis, and have resulted as follows:

Meningococcus has invariably produced an acid reaction in litmus-dextrose-serum-water, and in maltose-serum-water in the course of three or four days, without bringing about any apparent change in any of the others of the series of carbohydrates ordinarily used. Recently, before this society, Dr. Dunham has reported the same observation with reference to cultures of meningococcus isolated from spinal fluids.

The few strains of presumably true gonococci which we have thus tested have never produced any change which could be recognized with litmus. These cultures have been derived from cases of vulvo-vaginitis in children, from cases of suppurative oöphoritis, etc.

The cultures taken to be Micrococcus catarrhalis have varied in that some of them have not fermented any of these media, while others have acidified dextrose and maltose, and, furthermore, have coagulated saccharose-serum-water within twenty-four to forty-eight hours. These cultures included several kindly sent us by Dr. Libman from Mt. Sinai Hospital.

Meningococcus, therefore, gives in these media a definite reaction which serves to distinguish it from others of the group of morphologically similar Gram-decolorizing diplococci.

In accordance with this routine method the cultures isolated from these infected joints were tested upon the sugar media. In spite of other apparent resemblances the various strains described did not behave alike when grown upon these media. The cultures from Case I., obtained from two aspirations, produced a distinct acid reaction in litmus-dextrose-serum-water after four to five days, and in maltose-serum-water after a somewhat longer time. On the other hand, cultures from Cases II. and IV. produced no apparent change in the serum sugar media.

A complete review of the literature of this subject will not be attempted in this paper. A few reported cases, however, may be cited.

Wadsworth has reported the isolation of a Gram-decolorizing diplococcus from the knee-joint fluid in a case of scarlet fever.

In Mallory and Wright's "Pathological Technique," 3rd Edition, 1904, the following statement is made: "In proof of the necessity of cultures for confirming the identity of the gono-

coccus in certain instances, we may state that we have met with a Gram-decolorizing coccus in an arthritis of the knee, clinically of gonorrhœal origin, which, in cover-glass preparations from the exudate, was regarded as the gonococcus, but which was found not to be that organism by the study of it in cultures." No further description is given and we do not know whether the case has been reported in detail.

Gwyn, in the Johns Hopkins Hospital Bulletin for 1904, reports the finding of the diplococcus intracellularis of Weichselbaum, in the spinal fluid, in the circulating blood, and in the knee-joint fluid in a case of epidemic cerebro-spinal meningitis.

Evidence of pathogenic action of these micro-organisms in our cases is based only upon their presence in the joints and in the general circulation. These cases, however, illustrate the importance of the cultivation of the Gram-negative cocci, no matter where present—urethra, joint, etc.—through numerous sub-cultures before reporting them as gonococci. This fact has not been emphasized and its importance not generally recognized.

The association of joint affections arising from infection with cocci other than the genococcus with specific or non-specific urethritis, assumes further practical importance in judging of the benefits of treatment with anti-gonococcus serum, since, without bacteriological examination in these cases, no accurate conclusions can be drawn as to the value of such treatment.

DIAGNOSIS AND TREATMENT OF GASTRIC ULCER.*

By Charles E. Nammack, M. D.,

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Thirty years ago the term dyspepsia was readily offered by the physician as a complete diagnosis, and accepted by the patient as a satisfactory explanation of his symptoms. With the development of modern methods of study of diseases of the stomach, a tendency arose to refer every symptom to some definite anatomical lesion of the lining of the stomach, and the anxious layman began to expect that his family attendant would at once refer him to some stomach specialist of whose array of test tubes, stomach pumps, illuminators, stomach buckets, insufflators, electric buzzers and tickers, and even revolving scrubbing brushes vivid and wonderful accounts were published. For a time, there was danger that we might forget that we were not treating a sick stomach, but a sick man, and general examination was relegated to second place in our anxiety to make an elaborate diagnosis of the exact local pathological condition. Far be it from me to despise the help of chemistry, mechanics, or electricity in diagnosis, but experienced clinical observation yet has an important place at the bedside, and the laity are growing just a wee bit tired of some of the fads of specialism. In the elucidation of a stomach problem physical examination of the stomach itself and chemical examination of its contents should be the last steps. A careful study of the patient's family history, occupation, habits, variations in weight, and previous illnesses will often indicate at what point the wheels first left the track, and explain the bumps and jars and strains of the machinery since that time. Next, the present condition of the patient should be thoroughly investigated, especially the state of the heart, lungs and kidneys, including a twenty-four-hour estimation of the urinary excretion with special reference to indicanuria. The liver and intestinal dejections must

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next be studied, the latter with reference to their frequency, regularity, quantity and character. A blood count will be useful when marked anæmia exists, and helpful in suspected malignant disease, although marked blood changes are not conspicuous in the beginning of cancerous disease. The marked improvement in the blood condition following gastroenterostomy in stomach carcinoma shows indeed that the blood changes do not depend upon the cancer *per se*, but are rather the result of interference with assimilation. Finally, the nervous system must come in for its share of patient study, because it furnishes, in city life, the most frequent point of departure for failure of digestion. The pace at which we live in New York is best illustrated by the fact that we obey promptly, meekly and unprotestingly the order "Step lively, please!" which greets us several times a day, and, in our anxiety to get there, humbly "move forward" and hang on the strap. And the conditions of municipal travel only exemplify the conditions of municipal business and professional competition and social life. Even our amusements are strenuous.

Having completed our general survey of the patient, we are then, and not until then, ready for the physical examination of his stomach and of its contents. And just here I should like to interpolate two or three conclusions of A. L. Benedict, from a very valuable article abstracted in the *Medical Record* of December 17, 1898, on Conservatism in the Use of the Stomach Tube. "Don't discard external means of physical diagnosis because you have a stomach tube; don't imagine that the gastric douche will cure all the diseases of the stomach; you would laugh at a gynæcologist who held such a view about the vaginal douche. Don't let the patient learn to pass the tube himself; this rule holds for his benefit as well as yours." To which I should like to add one more: Don't depend exclusively upon the results of chemical examination of the stomach contents; they often lead experts astray. Bearing these things in mind, we are now ready to consider the topic of the evening as indicated by the title of this paper.

By the term gastric ulcer we are to understand a local necrosis resulting from the action of the gastric juice upon a focus whose resisting powers are for some reason diminished. This constitutes the typical peptic ulcer, or, as it is often called, simple, solitary, round, perforating, or "the ulcer of Cruveilhier." It differs clinically and histologically from the ulceration of stomach

cancer and from the various forms of ulcers common to other tissues as well as to the stomach lining. Chemical, mechanical, or toxic irritants, the acute infections, tuberculosis, syphilis, the blood diseases, and puerperal septicæmia may all produce localized suppuration of the gastric mucosa, but, with the exception of cancer, the concomitant symptoms will usually differentiate these. The literature of gastric ulcer has been very voluminous during the last two decades, yet little has been added to our real knowledge of the disease since the article of William H. Welch written twenty years ago. Some impressions of that period have since been corrected, however. We no longer consider the disease to be preponderant in young females between 20 and 30 years of age, and clinical experience has forced upon our attention the vast differences in management, prognosis and treatment between ulcers of the acute type and those of the recurrent and chronic type. Failure to sharply differentiate these types forms the groundwork of much that we hear and read about differences of opinion regarding medical versus surgical treatment. The real pathogenesis of the disease is unknown, although there have been many hypotheses. Pathological anatomy and the results of surgical operations on the living subject, however, both give us clear ideas of the course a gastric ulcer may follow. In some, only the mucous coat may be invaded, the base being formed by the submucosa, and these are the cases which heal spontaneously by cicatrization. In others the submucosa, as well as the muscular coat and the serous coat, may be perforated, the base of the ulcer being formed by a mass of connective tissue springing from the peritonæum, or from some adjoining organ. Peritonitis, either localized or general, may complicate the picture. In other cases, repair takes place by an increase of connective tissue in the stomach coats which, by subsequent contraction, gives rise to decided deformity of this organ, or to pyloric stenosis with its train of evil effects. This is the usual condition in frequently recurring ulcers or in those which have passed into a chronic condition, general peritoneal infection being more apt to occur when ulceration has been rapid or when it occurs at some spot which does not impinge against some other viscus. It has been asserted by some and denied by others, that gastric carcinoma frequently arises in the healing lesions of gastric ulcer.

The cardinal symptoms of gastric ulcer are unimpaired appetite with the patient afraid to eat because of the distress and

pain provoked by eating and occurring immediately after meals, tenderness localized to a small area, nausea and vomiting of very acid contents, hæmatemesis, and melæna. The last two symptoms may not be obvious, but the blood in vomitus and stool may require microscopical and chemical tests for its determination. Loss of weight and cachexia with secondary anæmia soon develop. Other complications are hæmorrhage; perforation, either free or with general peritonitis; subphrenic abscess; fistula between unsuccessful with certain cultures. Others, however, grew for stomach and adjacent organs; perigastritis ending in suppuration; perigastritis which may produce adhesions to various organs or displacements of the stomach or distention of the stomach; pressure on, or stricture of, bile ducts with jaundice; catarrh of gall bladder from adhesions; stenosis either of the pylorus, cardia, or the body of the stomach; severe gastralgia; tetany; acute or chronic pancreatitis; abscess of liver; chronic hepatitis; carcinoma, which may develop at the site of the ulcer and which occurs in five or six per cent. of the cases, according to Boas;[13] and great loss of flesh and strength ending in tuberculosis.[12]

In the typical cases, the combination of these symptoms is sufficiently striking to point the way to an easy diagnosis, but in the atypical cases diagnosis may be difficult or the disease only be discovered post-mortem. In 1,000 consecutive autopsies at Bellevue, only nine cases of gastric ulcer were found, and in but two of these had the diagnosis been made during life.[2] In 20,317 European autopsy records, Fenwick found about 1.5 per cent. of cases.[14] Chronic gastric ulcer is usually mistaken for cancer and I am convinced that lives have been lost by this mistake which might have been saved by timely surgical interference. In a recent personal case, occurring in a man 45 years old, irregular radiating pain, diffused tenderness, vomiting of stagnating long retained food, scanty coffee ground hæmatemesis, absence of HCl with presence of lactic acid, marked cachexia, and palpable tumor in the pyloric region, all led me to a diagnosis of cancer. Laparotomy showed tuberculous peritonitis and probable chronic tuberculous ulceration of the stomach. Gastroenterostomy by Dr. William T. Bull has prolonged this patient's life and greatly ameliorated his sufferings My diagnosis of cancer in this case was corroborated by some of the ablest general diagnosticians and stomach specialists in New

York, and operation had been unanimously agreed upon to relieve the pyloric stenosis.

Diagnosis.

In the average case of acute gastric ulcer, we must exclude hyperchlorhydria, acute catarrhal gastritis, gastralgia, cholelithiasis, cancer of the stomach, intercostal neuralgia accompanying digestive disorders, gastric crisis in locomotor ataxia and other diseases of the spinal cord, and duodenal ulcer.

In hyperchlorhydria the distress and pain are usually relieved by taking food, but return in an hour or two when the total acidity has risen. In acute catarrhal gastritis there is usually constitutional disturbance with fever, history of recent improper eating or drinking, loss of appetite, frequent violent vomiting, diffused pain and tenderness. Gastralgia occurring in young chlorotic women presents the greatest similarity to gastric ulcer and will sometimes be differentiated with difficulty. The suggestion of Murdoch[3] that the administration of orthoform will help in this difficulty has been verified in two of my cases since his publication. His explanation is that orthoform can relieve pain in the stomach only by coming in contact with a surface from which the mucous membrane has been removed, as it will not anæsthetize nerve endings protected by skin or mucous membrane. In gastralgia, pain may be independent of taking food; it is often relieved by firm pressure, and is rarely relieved by vomiting. Nevertheless, there are many doubtful cases, and it is safer to institute the proper treatment for gastric ulcer in them, as a diagnosis of purely functional gastralgia has often been overthrown by the occurrence of a profuse hæmatemesis.[4]

In a very considerable proportion of cases it may be entirely impossible to make a diagnosis between gastric or duodenal ulcer and gallstones, as the characteristic symptoms of either condition may be entirely absent.[4] The general diagnostic rule that the whole complexion of a case should be studied, rather than any fancied pathognomonic symptoms, applies here very forcibly. Experienced surgeons, like the Mayos, have often operated for gallstones and found duodenal ulcer.

The diagnosis between ulcer and cancer of the stomach is glibly recited in parallel columns in all the text-books. The recent activity of stomach surgery has destroyed our faith in the infallibility of these tables,[5] but they are of service in the typical

text-book cases, which cases, alas! are only met with in the text-books and not at the bedside. In diagnosis and treatment the mechanics of the stomach rather than its elaborate chemistry now holds first place.

Intercostal neuralgia is apt to be accompanied by digestive disorders which suggest gastric ulcer, but the tenderness is along the course of the nerve, and there is usually a history of other nervous manifestations, or of depressing influences affecting the nervous system.

The gastric crises occurring in spinal cord diseases are most frequently associated with locomotor ataxia, and although they occur in the preataxic stage, yet the coexistence of ocular disorders and fulgurating pains in the extremities may point the way to their recognition. In addition, they have as distinguishing features, sudden onset, atrocious severity, almost incessant vomiting, and continuance almost without omission for two or three days.

Duodenal ulcer is most often located in the upper two and one-half inches of the gut above the opening of the common duct, and depends upon the same main ætiological factors as gastric ulcer, *i. e.*, anæmia, hyperchlorhydria, and mechanical injury. Its symptomatology is almost identical, and its differentiation almost impossible. Pain an hour or more after taking food, very intense in character, and tenderness a little farther to the right than usual in gastric ulcer may indicate it. Repeated hæmorrhages through the intestine *only* favors duodenal, while increased HCl favors gastric ulcer. Duodenal is more liable to perforation than gastric ulcer, but protective adhesions are more apt to form because of its anatomical relations.

Given then our diagnosis of gastric ulcer, how shall we treat it? It is evident from what has gone before, that a great diversity in course and indications for treatment obtains in different cases and that each must come up for individual decision, rather than be included under any general proposition. The three leading ætiological factors are anæmia, hyperchlorhydria, and the various conditions, local or general, which hinder repair. Hence the three leading indications for treatment are to improve general nutrition, to diminish the excessive acidity of the gastric juice, and to put the ulcer at rest. In proportion to the ability of medical measures alone to meet these indications, will success attend their employment. And this brings us at once to the fact

that treatment must not only be addressed to the ulcer itself, but also to the consequences of the ulcer. Gastric ulcers may be divided clinically into acute and chronic. The acute form with its classical symptoms, pain, vomiting, local tenderness and hyperchlorhydria is readily recognized, and it is altogether probable that the vast majority of these cases will heal under medical treatment. In fact, study in the post-mortem room shows that four out of five simple ulcers heal during life.[14] Surgery has only to do with the complications of hæmorrhage and perforation. Slight hæmorrhage does not require surgical intervention, and even a single large hæmorrhage does not demand operation until medical measures have failed to control it. Death from hæmorrhage, according to Moullin,[7] occurred only once in 153 cases in women under 30 years of age. In Fenwick's cases, about 3 per cent. succumbed to this cause.[14] Recurring acute hæmorrhage and chronic hæmorrhage always demand surgical treatment. Perforation of an acute gastric ulcer also demands immediate operation as every hour's delay costs the patient eight per cent. of his chances of recovery,[8] and nothing is to be gained by waiting for the disappearance or amelioration of the shock which is usually present for some time after perforation has taken place. It is not necessary, before this society, to enter into details of medical treatment when this has been decided upon. Absolute rest, an ice bag, rectal feeding at the outset, followed by mouth administration of peptonized milk or other milk foods, expressed beef juice or Leube's beef solution when milk cannot be taken, and raw or soft boiled eggs, constitute the measures most favorable to repair of the ulcer. Sixty cases have been reported in which nutritive enemata were dispensed with, and the patients were given raw eggs with milk from the very first,[9] and raw scraped meat after the sixth day. Samuel W. Lambert[10] believes that it is safer to withhold stomach feeding, and depend upon nutritive enemata, for at least four days in the mild cases, and for a week or more in the severe cases. This is also the practice in Johns Hopkins Hospital.[11] Nutritive enemata must not be continued too long, as a patient may die from starvation despite their regular administration.

In deciding that we are dealing with an acute gastric ulcer, care must be exercised not to mistake an acute exacerbation of a chronic ulcer. Chronic gastric ulcers form the class of cases of "chronic dyspepsia" which go about from one physician to an-

other, and from one health resort to another, now gaining a little, again losing a lot, until pyloric stenosis or perigastric adhesions produce such changes in the mobility and contour of the stomach and accompanying chronic gastritis wreaks such havoc in its functionating power that the unfortunate victim must seek relief from suffering in the morphine habit. Happy, indeed, is he if he now falls into the hands of a surgeon who can procure for him gastric drainage, and American surgeons at the present day stand second to none in the accomplishment of this beneficial result. Surgery promises more in chronic than in acute gastric ulcer, because the former is solitary in about eighty-seven per cent. of the cases in which it occurs, while the acute variety exhibits a multiplicity in more than half (fifty-four per cent.).[14] Again, chronic ulcer exhibits a marked proclivity (seventy-six per cent., according to Fenwick) for the pyloric region of the stomach, where by extension and inflammation and adhesions it is apt to produce mechanical obstruction, or displacements, or distortion of the stomach. Adhesions, although meant by Nature to serve a useful purpose, prevent the contraction of the ulcer which is necessary to repair. They may also be the cause of hæmatemesis by erosion of some neighboring viscus. Or by rupture during sneezing, coughing, vomiting, or straining at stool, they may lead to an acute general peritonitis.

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THE MEDICAL TREATMENT OF DUODENAL AND GASTRIC ULCER.*

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To write a short discussion on the medical treatment of duodenal and gastric ulcer would have been a relatively simple task a few years ago; to-day it is far different and by no means easily performed. The difficulty arises in some degree because we realize more clearly than formerly that the course and prognosis of acute ulcer differ greatly from those of chronic ulcer, and especially because surgery has in a few years made such advances in the treatment of gastric ulcer that we must consider when medical treatment will suffice or when it is necessary for a surgeon to consider jointly what method of treatment will give the quickest and most permanent relief to the patient.

The medical treatment of duodenal ulcer being practically the same as for gastric ulcer, what is said for one will apply also for the other.

The acute and chronic gastric ulcer cannot always be clinically differentiated. Often what seems to be an acute ulcer is in reality an acute exacerbation of a chronic and long-standing ulceration. The medical treatment for the two conditions is practically the same, being modified, as medical treatment is for other conditions, to meet the varying indications presented by different individuals. There is no specific for gastric ulcer, and we must rely on the normal reparative processes of the body to effect a healing, realizing that these processes are going on in an environment unfavorable to rapid and complete healing, and often in a body far below its normal condition of health.

The conditions to be met with are hyperacidity and sometimes hypersecretion and stasis of food in the stomach; nausea, vomiting and pain arising from the morbid processes present in the

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gastric mucous membrane; the general conditions and symptoms of nervousness and anemia arising from the faulty nutrition of the body; and not infrequently the complications of hemorrhage, perforation and at times stenosis and distortion of the pylorus or other regions of the stomach.

Treatment.

Physical Rest—The first essential treatment is rest, complete rest for body and for stomach. This means rest in bed and rectal feeding, followed by continued rest in bed and milk diet. The length of time necessary to remain in bed varies from a week or two in acute and mild cases to several weeks in the cases of acute exacerbations of chronic ulcer. This rest cure does not mean the absolute isolation necessary in some cases of neurasthenia, but the patients must stay in bed and give up all the daily cares and worries of household and business. They may be entertained by reading and games and short visits from family and friends and may receive letters and write or dictate short replies. They must, however, remain in bed until the pain and tenderness following the ingestion of a mild but varied diet shall have disappeared. This will not average more than six weeks.

Rectal Feeding—As soon as the general bodily rest is assured we should begin the gastric rest by complete abstension from food into the stomach; only ice and small amounts of water should be permitted, and rectal feeding begun. Each morning there should be given a high lavage of the colon with about four quarts of warm normal saline solution. After a rest of an hour the regular nutrient enemata should be begun and given at four-hour intervals during the day. Three or at most four are sufficient for the twenty-four hours. Each enema should not exceed four ounces in amount, and may consist of eggs, milk, meat broths or meat powders, or a combination of these; but all enemata should contain only thoroughly predigested food, being thus prepared by pancreatic extracts and bicarbonate of soda. Two hours' predigestion is none too long for the preparation of these enemata. Alcohol is not an essential part of a nutrient enema and should not be given except for some special therapeutic reason, although it is a prominent ingredient of most proprietary peptone foods. About half a teaspoonful of table salt should be added to each enema. Some preparation of opium should

also be added if the enemata are not well borne, or if the rectum becomes irritable from frequent use, as the rectal feeding may thus be carried on longer than is possible without it.

After a couple of days the feeling of hunger from stomach starvation becomes blunted, but the feeling of thirst is troublesome, and besides the water by mouth, about 8 oz. of saline should be injected into the bowel about an hour after the last nutrient enema in the evening has been given. The daily colonic lavage will also supply some water, as some of it will be retained and absorbed, often half a pint or even more. The patient's sleep should not be disturbed, hence rectal feeding should be omitted during the night.

Even in mild cases rectal feeding should continue for three or four days, in more severe cases for a week or ten days, and in the exacerbations of chronic ulcer some authors recommend its continuance for twenty-one days. I believe we are more sure of a cure if even in the mildest cases we carry out for some period of time exclusive rectal feeding.

Gastric Feeding—Gastric feeding should be begun before the rectum grows intolerant; and, as we gradually increase the gastric, we should decrease the rectal feeding. Peptonized milk in small doses, given at long intervals, should be the first food given by the stomach. By increasing the amount and diminishing the intervals the average patient at the end of a week from the time gastric feeding was begun will be getting a quart of peptonized milk in twenty-four hours, with the rectal feeding discontinued. The next step is to increase the amount of peptonized milk until two quarts or more a day are given. The use of an exclusive milk diet is often objected to on the ground of personal idiosyncrasy, or because of the bitter taste of properly peptonized milk. But, as was emphasized two years ago by my brother, Dr. S. W. Lambert, when writing on this subject, if we will use the rules of modified milk as practiced in infant feeding, varying as may be necessary the amount of top milk or cream, lime water and water, and using milk sugar instead of cane sugar, it is possible to obtain a milk, raw or peptonized, which will agree with any idiosyncrasy. There is, however, no epicurean or short road to the proper and palatable peptonization of milk; and the peptonized milk which we must use, whether in the colon or stomach, requires the constant application of moderate heat for two hours for its preparation, and the product has a disagreeably bitter and

unappetizing taste. As soon as the patient is taking two quarts of peptonized milk a day we may begin to reduce the degree of peptonization; and usually during the third week the peptonizing process may be reduced to half an hour or twenty minutes' duration; and, by the end of another week, milk may be given raw, though perhaps still modified in strength.

As we reduce the peptonization we may add the cooked cereal gruels of barley, wheat, or oat flour as part of the diluent. During the fourth week, with the raw milk, we may add crust free bread, butter and eggs, but not hard boiled. It is well to add only one new thing at a meal that we may judge accurately if it agrees with the patient, and be ready to discontinue it if necessary. In the fifth week one may add potatoes (baked or boiled), rice (boiled), macaroni, custards, chicken and sweetbreads. In the final or sixth week of the cure, beef, mutton, green vegetables, light salad without vinegar, cooked fruits and simple puddings may be added.

During the period in bed the patients should receive daily alcohol spongings and baths and light massage to the arms, legs, chest and back, avoiding the abdomen, to improve the general condition. Iron in some non-irritating form, as the pyrophosphate, should be given if necessary. The average patient will be out of bed in the fifth week; in the milder cases this will occur earlier. Each day from this on should see the patient doing a little more, so that before long he may resume his ordinary existence.

The Lenhartz Protein Diet—Lenhartz recently has expressed his dissent from the abstinence diet and gives a protein diet from the first. Starting with two eggs and 6 oz. of milk on the first day, he increases the eggs one a day until eight are taken and continues at this number; the milk being simultaneously increased three ounces a day until a quart is taken and continued at this amount. The eggs and milk are given ice cold and in teaspoonful doses. On the third day two-thirds of an ounce of sugar is given, and this is gradually increased until on the ninth day 1.6 oz. are given, and this amount is continued. On the sixth day an ounce of raw scraped beef is given, double this amount on the next day, and so continued. On the seventh day 3.3 oz. of milk cooked with rice are added and gradually increased to ten ounces on the eleventh day. On the eighth day two-thirds of an ounce of zwiebach is added and gradually in-

creased until 3.3 oz. are given on the fourteenth day. On the tenth day 1.6 oz. of raw ham are given and continued daily in this amount. Butter is also added on the tenth day, giving two-thirds of an ounce, and double this amount on the next day and continued. Lenhartz reports 60 cases treated by this method with gratifying results.

Danger of Relapse—Whatever the diet and method of cure we pursue, after we consider our patients cured we must always remember the danger of relapse or a recurrence of the ulcer; and we must impress on them the necessity for a prophylactic diet and mode of life which we must insist they observe for a long time. These patients must avoid frequent eating and drinking and at all times the overdistension of the stomach with food. They must eat slowly and masticate their food thoroughly. No irritating or hard foods, such as bread crusts, fruit seeds, tough vegetable or meat fibers must be given the chance to irritate mechanically the gastric mucous membrane. Alcohol in all its forms, spiced and highly cooked sauces and condiments and even excess of common salt must be avoided lest there be a chemical irritation in the stomach. As recommended by Shattuck, from three to six months after they seem well they should take before each meal from one-half to two teaspoonfuls of bismuth subnitrate. Physical and mental overfatigue must be avoided and no journeys should be undertaken on which they cannot control their diet and mode of life.

Drug Measures—Two therapeutic measures with drugs used in connection with the dietary régime deserve special mention. One is the bismuth cure, as described by Fleiner, the other the olive oil cure of Cohnheim.

1. Fleiner's Method: Wash out the fasting stomach each morning until the water returns clear and is free from all acid reaction; then introduce through the tube six ounces of water in which 2.5 to 5 drams of subnitrate of bismuth is evenly suspended. As soon as the tube is withdrawn the patient should lie down and rest quietly in such a position that the bismuth will gravitate toward the supposed site of the ulcer. After half an hour's rest the patient should be given his breakfast. The use of the bismuth should begin with the gastric feeding and continue for two weeks. Fleiner claims for this bismuth cure both an antiseptic and astringent action, besides a reduction of the hyperacidity and a mechanical protective effect of the large dose. The

result of such treatment given daily should be immediately apparent in a diminution of the stomach distress and a cessation of the pain, nausea and vomiting.

Ewald has suggested the use of the insoluble carbonate of lime instead of the bismuth; and Pariser has obtained excellent results by the use of a mixture of chalk, talcum and magnesium in place of the bismuth.

2. Cohnheim's Method: Introduce olive oil through the tube into the fasting stomach after a moderate lavage, before breakfast, in quantities of from two to four ounces; or give it before the three principal meals, without the use of the tube, in doses of one to two ounces. Cohnheim claims that the oil forms a protective coating to the ulcer, relieves the pain and vomiting and tendency to pyloric spasm, allows the patient to be fed, itself acts as a food and, finally, that it checks the secretion of acid. From his reports it would seem that the milder cases can be treated under this oil regimen by simply adding a regulated diet without any rest cure.

Krausz has recently recommended a mixture of 75 grains of sodium bicarbonate in three ounces of olive oil to be given in tablespoon doses before each feeding.

Objections—The objections urged against the bismuth and oil cure are the use of the tube in gastric ulcer. The bismuth and oil are best given by a tube, but they can and often have been given directly by the mouth. The question of the use of the stomach tube in gastric ulcer is one not always easily decided; but, if care is observed not to distend the stomach and too great pressure is prevented by limiting the height of the funnel to the head of the patient, the risk of perforation of the ulcer is slight.

But in those cases in which there has been recent hemorrhage, or if even slight hemorrhage follows the lavage, the tube should not be used. Two cases of fatal hemorrhage have been recorded, due, it was believed, solely to gastric lavage.

Other objections to the bismuth cure of constipation and discoloration of the stools, preventing the observation of malæna from slight hemorrhages, do not hold good. Large doses of bismuth do not seem to constipate, the guaiac test for blood in the feces is not obscured by bismuth and is so easily performed that it should be a routine procedure carried out at least every few days.

Silver Nitrate and Iron Perchlorid—The principal drugs used for their astringent action and in the hope of stimulating the ulcerated surface to cicatrize are the perchlorid of iron and nitrate of silver; of these the silver is by far the most useful. To insure all the silver reaching the stomach in an active condition the solution must be given through a tube. But, if the use of the tube is undesirable, the nitrate of silver can be given in pill form in .25 to .5 grain doses, or, better still, the same dose in solution with 5 to 10 grains of bicarbonate of soda given on an empty stomach three or four times a day. The nitrate of silver acts both as an astringent and as an antacid; for, if it is precipitated as an insoluble chlorid, it uses up in the process some of the hydrochloric acid in the stomach. Boas recommends a solution of silver nitrate, four grains to four ounces of water, a tablespoonful to be taken on an empty stomach three times a day. After one bottle of this has been taken, five grains of silver nitrate to four ounces of water are given and two bottles of this strength used. Then seven grains to four ounces are given and two bottles of this taken. The nausea sometimes occurring from this medication may be corrected by peppermint, and the slight diarrhea seen at times in the beginning of the silver cure will cease spontaneously during its further administration.

The Alkalies—Simple antacid and some laxative action is all that is usually ascribed to the alkalies, and often Carlsbad salts are given the first thing in the morning as routine treatment; but Myer, quoting Binet in a recent communication, attributes still further action to calcium phosphate, sodium bicarbonate and magnesium which, he claims, increase the gastric secretion through stimulation. This action continues up to the point of saturation of the hydrochloric acid in all three cases, but in different degrees, the action being slight after sodium bicarbonate and very marked after magnesium. Sodium bicarbonate also stimulates the motility of the stomach through the liberation of carbon dioxid. It acts on the sensibility only when pain develops a long time after eating. And such pains do not always point to hyperacidity. Often when the pain is greatest the stomach contains no free hydrochloric acid or less than is found in a test meal which has caused no pain. These pains, therefore, are dispelled more promptly by sodium bicarbonate, which neutralizes but little acid, than by magnesium and calcium, which neutralize more acid. Binet believes that this pain which develops some time after the

ingestion of food is due to pyloric spasms, which are relieved by sodium bicarbonate, because of its tendency to empty the stomach through the stimulation of the motor function.

Symptoms and Complications—In the treatment of any given case of gastric ulcer there is often a predominance of some symptom or the occurrence of some complication which demands special treatment. The nausea and vomiting and nervous symptoms are often allayed by the treatment of the ulcer itself by diet, rest and the bismuth or oil as already described. If they recur during dietary treatment, often a change back to a simpler diet will suffice to allay them again. Antiemetics are certainly not contraindicated and are often of benefit. The pain may disappear without further medication during the gastric rest with rectal feeding, or it is often allayed by the silver nitrate and sodium bicarbonate; if not, codein, cocain or anesthesin may prove effective; the relief by orthoform in my experience has been of but very short duration. Morphin will relieve, but tends to increase the gastric acidity. Hyperacidity, whether it is a cause or an effect of ulcer, is usually present and often requires attention. Frequently the simple neutralization by sodium bicarbonate is sufficient. The use of bismuth, oil and fatty foods and irrigation by silver nitrate secure the same results. Atropin or belladonna has been used successfully to control or check the secretion of the acid.

The serious complications of ulcer are perforation and hemorrhage. Perforation is a surgical condition, and one cannot emphasize too strongly the urgent necessity of an operation at the earliest possible moment after the perforation has occurred. Slight hemorrhages not infrequently occur in the course of treatment of ulcer, and demand no deviation from the lines of treatment already laid down. Hemorrhage is often the first indication that a patient has an ulcer and when the amount of blood lost can be measured by ounces immediate treatment with a period of initial gastric starvation should be instituted. If the hemorrhage, even though it has been severe, is not repeated, the regular routine treatment should go on. If a considerable hemorrhage is repeated, or if there are repeated small hemorrhages sufficient to reduce the vitality of the patient, medical and surgical treatment should be discussed immediately and the physician should not look on the surgeon as the last resort. The medical measures recommended for hemorrhage are absolute rest in bed, morphine if necessary to obtain physical and mental quietude, the swallow-

ing of ice, cold application on the abdomen, subcutaneous injections of ergot, one or two teaspoonfuls of turpentine mixed with the white of an egg and thirty drops more after an hour if necessary, and solutions of adrenalin chlorid left in the stomach by a tube, clots in the stomach being first washed out and the stomach allowed to contract.

After a severe hemorrhage the question arises whether a saline infusion should be tried. If the heart can carry on the circulation without it, it should not be given; the blood will tend to coagulate and plug the bleeding point better without it, as the infusion may wash out the plug. If it seems necessary it may be given, but only enough to enable the circulation to go on and one should be satisfied with a relatively poor pulse. Subcutaneous injections of normal saline are better, and often sufficient fluid can be absorbed by injecting 8 to 10 oz. of saline into the colon and repeating this procedure a number of times at two-hour intervals. Vigorous cardiac stimulation is certainly contraindicated.

Results.

This must suffice for the medical treatment. Let us now consider its results. Taking the statistics recently published by Dr. Geo. G. Sears* of this city, and adding Leube's statistics, we have 1,203 cases of gastric ulcer in which the immediate results of treatment were 88.3 per cent. cured or improved. Considering the end-results after four or five years in 647 patients, of whom such statistics are available, there is a further history of 14.2 per cent. to 37 per cent. of relapses, some patients remaining well after the relapse, 5.2 per cent. were unimproved and 5.8 per cent. died. And, finally, we have 52 per cent. as cured, 40.8 per cent. as failures, 3.6 per cent. dead of ulcer and the rest dead of intercurrent diseases. These are not encouraging statistics, but there are undoubtedly among the failures many cases which to-day would without question be sent to the surgeon. There is no question to-day that, besides perforation, all cases of pyloric stenosis and distortion of the stomach from cicatrices or perigastric adhesions belong to surgery. If we could but know how many of the medical failures belonged to these surgical classes, we could form a more accurate judgment of the value of medical or surgical treatment in those that remained.

* Boston Med. and Surg. Journal, vol. cliii., p. 369.

Leaving these statistics as they stand, physicians are to-day waiting for surgical statistics of end-results from gastric surgery. Surgical literature seems more concerned with its immediate results and the perfection of its technic than in reporting the value to the patient of the operation performed. Leaving out of medical consideration, as has just been said, all cases of mechanical obstruction and distortion, there remains to be considered acute and chronic ulcer and the question of surgical interference in hemorrhage. In young individuals, suffering in all probability from acute ulcers, medical treatment has obtained its best results; and could we separate these cases from the general mass of statistics, it is my belief we would find a high percentage of actual cures.

In reviewing the results of gastroenterostomy in these cases, Clairment† mentions a case of perforation and death and seven fatal hemorrhages following the operation and concludes that palliative gastroenterostomy for open ulcers gives poor lasting results and does not protect the patient from the dangers of perforation and hemorrhage. If the operation does not protect against these two dangers, it is certainly wise to wait until these dangers arise and to consider acute ulcers as belonging to medical treatment until life is threatened by the acute complication. These patients are in more danger from perforation than from hemorrhage; and, even after severe hemorrhage has occurred, medical treatment will save a greater number than surgical.

In what conditions of hemorrhage, then, can the physician turn to the surgeon with a fair promise of improving the chance for life of his patient? The impossibility of locating the ulcer and bleeding point before operation, frequently even at operation, and the high mortality of operations performed for severe hemorrhages convince us that we are not just to our patients nor to the surgeon if we consider surgery as the last desperate chance. We will make few errors if we accept Leube's indications:

1. Repeated, little, unceasing hemorrhages, sapping the vitality of the patient, absolutely indicate early surgical interference, and all the more so if stasis is also present.

2. A simple profuse hemorrhage is not a surgical indication. But if it is repeated, an operation is relatively not absolutely indicated. An operation is only indicated if the pulse and general condition of the patient justify it.

† Arch. f. Klin. Chir., vol. lxxvi., 1905.

In chronic ulcer, the condition in which most medical failures occur, does surgery offer a better chance of cure than medicine? In skilled hands the mortality of gastric surgery for ulcer to-day is about the same as in medical treatment without surgery. But, until the best operation and its technic is more generally agreed on, so that the skill of a few is more widely diffused and more statistics of end-results of operations are published, physicians cannot enthusiastically adopt a sugical point of view. At present we are constrained to believe that all chronic ulcers should first be treated by a most careful and strict medical regimen; if that fails and the patients cannot live in reasonable comfort or are debarred from the occupations necessary for their livelihood they should then undergo surgical procedures.

A CASE OF SYPHILIS OF THE STOMACH.*

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Although a considerable number of cases of syphilis of the stomach have been reported, there are comparatively few of these which bear rigid criticism, and the condition must still be considered a medical rarity. It is for this reason that we feel justified in reporting a case in which the diagnosis, as revealed by the necropsy and the subsequent histological study, appears to be beyond question.

The patient was a male, aged 32, German by birth, cook by occupation, admitted to the service of Dr. George Roe Lockwood, at Bellevue Hospital. Because of his precarious condition when admitted, no attempt was made to obtain a comprehensive history, and, unfortunately, no record of previous venereal infection was elicited. It was ascertained only that, for the past five months, he had been complaining at times of gastric distress after eating. At no time did this amount to actual pain. He had never vomited prior to the day of admission, when, without warning or apparent cause, he suddenly vomited a large amount of bright red blood. He became faint and weak, and was brought at once to the hospital.

Physical examination upon admission showed a poorly nourished individual of medium stature. There was marked anæmia; the skin was of a waxy pallor; the mucous membranes blanched. Pulse was 90, regular, of fair force and size, and low tension. Radial artery showed slight diffuse thickening. The heart was negative, save for localized blowing systolic murmurs over the base—probably anæmic. Lungs were negative. Abdomen was rounded, symmetrically prominent in the epigastrium, held somewhat tensely upon the left side, elsewhere soft. Slight tenderness on moderate pressure just below ensiform. No rigidity of either costal arch. Note over epigastrium was flat; elsewhere

* Presented in abstract at a meeting of the Pathological Society.

tympanitic. Lower border of stomach not determined. Liver dullness began at fourth space; flatness at fifth space, extending to free border of ribs. Edge not palpable. Extremities were warm. There was marked serration of the tibial crests.

Blood Examination.

Erythrocytes	1,600,000
Haemoglobin	20%
Leucocytes	6,400
Polymorphonuclears	76%
Lymphocytes	20%
Eosinophiles	4%

Smear shows no nucleated reds; moderate poikilocytosis; slight anisocytosis; no granular degeneration or polychromatophilia.

Urinary examination showed a slight trace of albumen and numerous hyaline and granular casts.

A diagnosis of gastric ulcer was made, and accordingly all food by mouth withheld. Large doses of morphine were given, together with hypodermic injections of saline solution to relieve thirst.

In spite of these measures the patient died after three days from repeated and copious gastric hæmorrhages. In all, about 75 ounces of blood were vomited. Examination of the blood the day before death showed normoblasts to be present in considerable numbers.

Autopsy—Performed February 12, 1906, twenty-four hours after death, through abdominal incision:

General Appearance—Poorly-nourished male, about 40; marked blanching of skin and mucous membranes: no enlargement of superficial glands; no scars on external genitals; panniculus scant; muscles pale; peritoneal cavity and appendix normal; liver reaches about two fingers below costal border; diaphragm fourth space right; fifth left; right lung adherent at apex; left lung free from adhesions; pericardial cavity normal.

Heart—Somewhat small; valves and orifices normal. Myocardium somewhat brownish. The aorta, from its inception to its bifurcation, is the seat of marked atheromatous change. In the ascending and transverse portion of the arch of the aorta, the intima is swollen, and is marked by linear fissures or irregular depressions, and by more or less numerous areas of fatty degeneration. Thoracic and abdominal aorta is the seat of numerous

old calcific plates. The peripheral vessels show a diffuse sclerosis.

Lungs—Pale; somewhat emphysematous and markedly œdematous. There is an old fibrous tuberculous process at the right apex. Bronchial lymph nodes are small and anthracotic.

Liver—Weighs 3 lbs., 14 oz.; surface is marked by irregular depressions, varying in size and depth; left lobe is small, deeply lobulated by constricting bands and scars. On section throughout the liver substance are numerous nodules, varying in size from ½ to 2 cm. These consist for the most part of firm, whitish tissue, and are surrounded by translucent connective tissue; others are irregular in outline, show no definite encapsulation, and extend into the surrounding liver tissue in the form of whitish bands of scar tissue. The substance of the liver is further traversed by irregular fibrous strands, originating in part from the capsule, and in part from the periphery of the nodule. The parenchymal tissue is brownish-gray in color. Lobules are poorly defined. There is a general increase in the perilobular connective tissue. Gall bladder is distended with tarry bile. Ducts are patent. There are fibrous adhesions between the diaphragm and the superior surface of the liver.

Spleen—Weighs 1 lb., 8 oz.; approximately three times normal volume; capsule is tense, and slightly thickened. On section, color is pale. The trabeculæ unduly prominent. Consistency of the organ is firm.

Pancreas—Large, pale and very firm.

Adrenals—Normal.

Kidneys—About normal size; capsule strips, leaving a smooth surface; color is somewhat pale; cortex normal width; markings indistinct; pelves and ureters normal. In the cortex of the right kidney is a small, whitish, firm nodule.

Bladder—Normal.

Prostate—Slightly large. On section shows a yellow mottling.

Testes—Normal.

Œsophagus—Œsophageal veins are visible, but not markedly varicosed.

Stomach—Contains a recent blood clot, about the size of an orange. The mucous membrane of the lesser curvature is the seat of an extensive serpiginous ulceration, involving an irregular area approximately 6 x 5 cm. This is situated on the posterior wall, extending across the lesser curvature for a distance of sev-

eral cm. The ulcer has the following characteristics: The edges are irregularly sinuous; slightly raised; not indurated. The floor of the ulcer is formed by the submucosa, is clean, and not covered by slough. Within the confines of the ulcer, islands of intact mucosa remain. About 2 cm. from the left border of the cardiac orifice, just within the margin of the ulcer, are seen the eroded stumps of two vessels, which project several mm. above the submucosa. About 3 cm. from the pyloric ring, along the greater curvature, is a small polypoid outgrowth, and adjacent to this, a superficial irregular ulcer, about 2 cm. in diameter, having the general characteristics of the larger one just described. In the floor of this a small, whitish nodule, about 2 mm. in height. Along the greater curvature, about 10 cm. from the pylorus, a small superficial erosion of the mucosa.

The wall of the stomach, from the pyloric ring to beyond the limits of the ulcer, shows marked thickening. This is due to the replacement of the submucosa principally, by firm, whitish infiltrating tissue. On cross-section, this measures about 5 mm.; the muscular layer about 2 mm. This new tissue extends into the intermuscular septa. The mucous membrane outside of the erosion is everywhere somewhat swollen; in places mammillonated, and covered with tenaceous mucus. The peritoneal surface of the stomach is normal.

Duodenum, Small and Large Intestines—Apparently normal. Small and large intestines contain black, tarry material, evidently decomposed blood.

Pancreatic, Perigastric and Mesenteric Lymph Nodes—All are considerably enlarged, ranging from 1 to 3 cm. in diameter. Section through these lymph nodes shows circular areas of firm, whitish tissue between which are seen recent reddish hemorrhagic extravasations, which appears above the surface.

Head and Brain—Not examined. (Autopsy performed through abdominal incision.)

Anatomical Diagnosis—Tertiary syphilis; cirrhosis of liver, syphilitic; multiple gummata of liver and mesenteric lymph nodes; ulcer of stomach, syphilitic; general arterio-sclerosis.

Microscopic Examination—Stomach—Tissue was taken for examination from various portions of the organ, fixed in formol-Müller, stained by iron-hæmatoxylin-eosin, Van Gieson's connective tissue stain, Weigert's elastic fibre stain, and Unna's polychrome methylene blue for the demonstration of mast-cells.

In the region of the pylorus, the mucosa, which is intact, measures approximately 2 mm.; the submucosa 1 mm.; the muscularis is slightly hypertrophied, measuring 5 mm. The mucous membrane shows a moderate diffuse adenomatous hyperplasia. The muscular fibres of the muscularis mucosa appear swollen and vacuolated, their nuclei large and poor in chromatin. The submucosa is œdematous; the connective tissue fibres swollen and hyaline. Many of the arteries and veins show a diffuse sclerosis with moderate proliferation of the intima and perithelium. There are a few small perivascular aggregations of small round cells. The fibres of the muscular coat are irregular in contour, swollen, and stain poorly. On cross-section, many are unstained, vacuolated or necrotic. The nuclei are swollen and stain faintly. Many divisional forms are seen. In certain areas the fibres have undergone a diffuse hyaline change; the nuclei of these fibres are frequently pycnotic. There is some increase of connective tissue between the bundles of muscular fibres. The intermuscular capillaries show swelling of the endothelium. The cells of Auerbach's plexus seem of unusually large size. With Unna's polychrome stain, a few scattered mast-cells are found in the submucosa, chiefly in the peri-vascular lymph spaces.

Sections taken through the edge of the ulcer show the following features. The epithelium at the margin is partially exfoliated, lying in or upon a highly cellular stroma, composed of fibroblasts, plasma cells and very numerous lymphoid cells. The floor of the ulcer, where the mucosa is entirely wanting, is covered by a cellular exudate consisting of cast-off and necrotic epithelial cells, numerous small round cells, epithelioid cells, a few polynuclears, granular detritus and mucus. Beneath this may be distinguished the fibres of the muscularis mucosæ, crowded apart by an infiltrating exudate of lymphoid and plasma cells. The submucosa in this region shows great thickening, measuring 3-4 mm. It is composed largely of œdematous, hyaline connective tissue fibrils, staining an intense carmine with Van Gieson's stain. Here and there are collections of lymphoid cells, arranged chiefly about the vessels. Cellular elements between the connective tissue fibres are comparatively scanty and consist principally of epithelioid cells with pale vesicular nuclei. The vessels do not show extreme changes. Some are practically normal. Others show a moderate thickening and fibrosis of all the coats, with swelling and proliferation of the endo and perithelium. A few of the smaller

arteries alone show an advanced obliterating endarteritis. Muscular coat is characterized by necrotic changes in the fibres similar to those noted in the pyloric region.

Horizontal sections were made at different levels through the eroded vessels at the margin of the ulcer. In all sections the wall of the vessels is replaced by a mass of necrotic tissue, consisting of areas of coagulation necrosis in and about which are very numerous epithelioid cells, lymphoid cells and nuclear fragments. A few muscular fibres may be found imbedded in cellular exudate. All the coats of the vessels are equally involved so that it is impossible to decide whether the structures affected are arteries or veins. The intima is entirely lost. The lumen of the vessels contain masses of red globules, blood plates, granular fibrin and exfoliated endothelium. By Weigert's elastic tissue stain, the subendothelial fibrils appear swollen and in places forced apart by the cellular exudate. In that portion of the new tissue representing the media and adventitia, elastic fibres are few and appear curled and twisted upon themselves.

The liver contains numerous irregular areas of coagulation necrosis. These are surrounded by a narrow zone of epithelioid cells, beyond which the tissue is extensively infiltrated with small round cells. The caseous centres contain a few pycnotic or fragmented nuclei; the innermost epithelioid cells show a distinct radial arrangement. The nuclei stain rather deeply, and have an elongated or pyriform contour, with pointed extremities directed centrally. Situated in this zone are a few very large giant cells, with twenty or more oval nuclei at the periphery. The lymphoid cells about the epithelioid zone extend in all directions into the liver tissue, infiltrating the cords of liver cells.

There is also an irregular cirrhosis taking its origin from the capsule and the periportal spaces. The new connective tissue is in places cellular; in others, hyaline. There are scattered collections of lymphoid cells, especially beneath the capsule, and in the periportals. There are no marked changes in the periportal vessels or central veins, save in the vicinity of the caseous areas, where there is very evident endo- and periarteritis. Elsewhere, there are found vessels in which the endothelium is stripped off, and lies free within the lumen; or in which the cells are somewhat swollen and proliferating. The parenchymal cells in the regions uninvaded by the granulomatous processes show no marked lesions.

Mesenteric, Pancreatic and Perigastric Lymph-Nodes all show similar lesions. There are irregular areas of coagulation necrosis, and diffuse cellular infiltrations with plasma cells, epithelioid and small round cells. Large giant cells are frequent. The reticulum of the lymph-spaces shows swelling and hyaline degeneration. The endothelial cells lining them are swollen. The lymphoid follicles unaffected by the process appear normal. Throughout the glands, especially adjacent to the necrotic areas, are scattered hemorrhages.*

Aorta—Sections were taken through the atheromatous areas in the thoracic region. The endothelium is everywhere intact, showing neither proliferation nor exfoliation. The subendothelial tissue is greatly thickened, in places equalling the muscular coat in width. It consists of homogeneous or indistinctly laminated connective tissue in which there are very few cellular elements. Immediately beneath the endothelium, in a few places, the tissue appears necrotic and granular, and is infiltrated with lymphoid cells.

The media shows diffuse areas of necrosis, where the muscle fibres are replaced by granular material, staining diffusely with eosin. With Weigert's elastic stain, the elastic fibres in these areas appears broken up, and have lost their normal regular arrangement.

The adventitia shows hyaline degeneration of the connective tissue fibres. There are numerous areas of round cell infiltration, especially about the vasæ vasorum. The latter show a moderate grade of mesarteritis.

Kidneys—The epithelium of the convoluted tubules is swollen and granular. The lumina of the tubules contain granular detritus. The glomeruli appear normal. The stroma shows no increase in connective tissue. Sections from *myocardium, pancreas, spleen, testis, prostate and adrenals* show no noteworthy changes in these organs.

It would seem obvious that those cases should be discredited, in which the diagnosis rests solely upon the beneficial effects of mercury or iodides, and is uncontrolled by autopsy findings and thorough histological examination. In spite of this, Dieulafoy,[1] Einhorn[2] and others have advanced the opinion that the therapeutic

* Pieces of tissue from the liver and mesenteric lymph nodes, after prolonged fixation in Kaiserling's solution, were stained according to Levaditi's method for the Spirocheta Pallida. A few atypical moniliform threads were found, but no typical spirals.

test is sufficiently specific to justify a diagnosis of gastric syphilis. If this were so, the number of cases would indeed be multiplied. We know, however, from the frequency of cicatrices in the stomach as found at post-mortems, that the ordinary peptic ulcer shows a strong tendency to heal. A simple ulcer showing no histological evidence of syphilitic origin, has been found at autopsy, associated with very advanced tertiary luetic lesions of other viscera. Again, in cases of grave syphilitic disease, the disappearance of vague gastric symptoms under appropriate treatment should not be taken to prove that there have been definite syphilitic lesions of the stomach, since the gastric symptoms might be attributed with equal justness to the anæmia and low general condition of the patient.

A number of writers have reported cases in which the diagnosis was based solely upon clinical evidence, namely, Andral,[3] Galliard,[4] Gouzot,[5] Dieulafoy,[1] Mackay,[6] Einhorn,[2] Julien,[7] Stockton,[8] and Hoover[9]. Without further analysis, we may exclude these cases from serious consideration as "not proven." As an example of the loose way in which the diagnosis has been made in some of these cases, we may epitomize one of the three cases of "gastric syphilis" reported by Hoover. The patient was a man of 50 years, who had recovered from his tertiary lesions 25 years previously. His symptoms were vomiting, constant epigastric pain, and emaciation. There was temporary improvement under iodides, followed by death. No autopsy was held. The possibility of carcinoma seems not to have occurred to the author.

A number of cases have been recorded in which the autopsy has been inconclusive or not accompanied by histological examinations, or in which the records of the histological examinations were unconvincing. Of the earlier cases, we must discredit those of Cruveilher,[10] Fauvel,[11] Leudet,[12] Capozzi[13] and Lancereaux[14] in so far as the diagnosis was based entirely upon the gross appearance of the lesions and the presence of syphilitic changes in other viscera. Wagner[15] described a syphiloma of the stomach, which he found at autopsy together with a syphiloma of the larynx and other syphilitic lesions elsewhere. In the stomach there was a sharply circumscribed oval thickening of the submucosa, muscularis, and serosa, about one inch in length, situated on the posterior wall. Also two small circumscribed areas midway between cardia and pylorus, grayish-white punctæ lying in the mucosa itself. Wagner states that the histological description is given

elsewhere, but we were unable to find it. Chiari,[17] in his review of the cases, also says that he searched for it in vain, and therefore rejects the case as incomplete.

The case reported by Weichselbaum[16] has been accepted by Chiari,[17] Stolper[18] and Flexner,[19] probably because of the coincidence of marked luetic lesions of the nose, pharynx, larynx, cranium and liver. The patient was a man of 25 years, who died of erysipelas. There were found in the stomach two ulcers; one, 12 mm. in length, situated on the posterior wall above the greater curvature; a second, the size of a millet seed, near it. The base of these ulcers was formed by hard cicatricial tissue to which the edges of the ulcer were firmly adherent. There was also a radiating scar about 2 cm. in size.

There is no record of a histological examination in this case. As the gross appearance of the lesions is not distinctive of syphilis, and as the possibility of a cicatrizing peptic ulcer cannot be excluded, it seems to us that this case also should be rejected as "not proven."

Klebs,[20] in 1869, described a lesion of the stomach which he considered to be of syphilitic origin. On the posterior wall near the lesser curvature the mucous membrane was ulcerated over an area the size of a franc piece. There was diffuse thickening of the submucosa and muscularis. The peritoneum was tendinous in appearance and showed yellowish fatty masses over the base of the ulcer.

Although this case has been considered authentic by Chiari and Flexner, it hardly meets the rigid criteria we have adopted, in that the diagnosis was unconfirmed by microscopic examination of the tissue. The gross appearance of the lesion is suggestive, but not conclusive.

Lafleur,[21] in 1903, reported a case in which the diagnosis was made at operation. The patient was a man 39 years of age, who had become infected five years previously, showing slight secondary and no tertiary symptoms. His gastric symptoms before operation were flatulency, fullness in the epigastrium, and occasional vomiting after ingestion of food. There was absence of free H Cl in the stomach contents. Lafleur's description of the lesion found at operation is about as follows: Extending from a point 2½ inches from the pylorus over to the cardia, the stomach wall felt thickened; the surface was of a deeper pink and showed scattered yellow "puckerings." No enlarged glands were

found. The liver along its edge presented a few nodules containing gritty calcific matter. On opening the stomach the wall was found thickened to 1 or 1½ cm. There was little bleeding. The everted surface showed a complete denudation of the mucosa extending completely around the stomach at the pyloric end of the incision and four inches toward the cardiac end. Here and there were small islets of mucous membrane having a rough coxcomb appearance and a purplish tint. The edges of the ulcer were well-defined, serpiginous, abrupt, slightly heaped-up and undermined. The floor of the ulcer was smooth—neither caseous nor necrotic, pink-red and bloodless. In the thickened area there had been some cicatrization, producing an hourglass constriction 2-3 inches from the pylorus. The microscopical examination by Dr. Wooley of the tissue removed at operation is reported as follows: "The tissue from the base suggested malignancy, for there were small masses of epithelial cells surrounded by a fibrous stroma. But the edges of the ulcer were simply fibrous tissue and muscle, the former in excess, and there was no marked infiltration. The base was markedly inflammatory, not malignant."

In criticism of this report, we may quote that the pathologist virtually contradicts himself in the first and last sentence. However, though incomplete and contradictory, there is nothing in the histological description that suggests syphilis, and the thought at once occurs that we are dealing with a beginning carcinoma on the base of an old ulcer. The further course of the disease is not stated, but in the absence of other definite evidences of syphilis the diagnosis is open to serious question, and the case should not be recorded as one of gastric syphilis.

There remains a small number of cases of congenital and acquired syphilis of the stomach, in which the diagnosis rests upon sound pathological evidence.

Cornil and Ranvier[22] have reported a case showing three gummata—5, 3 and 2 cm. in diameter, situated along the lesser curvature near the pylorus. The overlying mucosa was intact but adherent. On section the submucosa was fibrous, dense and yellow-gray in color, and had a thickness of 8-12 mm. There was slight hypertrophy of the muscularis. Histological examination showed the tumors to consist of vascular granulation tissue, with numerous lymphoid and embryonal connective tissue cells. Lesions of the blood-vessels are not mentioned. The diagnosis was made of fibrous gummata.

Birsch-Hirschfeld records two cases of acquired and one of congenital gastric syphilis. The first case of acquired syphilis was in a male 35 years of age, who had become infected four years previously. No further clinical history is given. Autopsy showed a large ulcer in the lower portion of the œsophagus extending to the stomach. The base was partly flat, partly overlaid with cheesy masses. The edges were in places fibrous and indurated, in others showed the gross and microscopic characters of gummatous formation. There were typical syphilitic lesions in the bronchial and mediastinal glands, radiating cicatrices in the liver, and gummatous plaques in the upper portion of the small intestine. The second case was a male, 45 years old, syphilis acquired six years previously. At autopsy, large gumma of the liver was found. On the anterior wall of the stomach, near the pyloric region, a hard, oval area, 8 cm. in length, superficial ulcer in central portion. Floor smooth and yellowish in color. Base and raised edges of firm cicatricial tissue which replaced the submucosa and infiltrated the muscularis. Microscopical examination showed this to be a cicatrizing granulation tissue, with obliterating endarteritis.

Chiari,[17] in 1891, thoroughly reviewed the previous cases, accepting as positive those of Klebs,[20] Cornil[22] and Ranvier, Weichselbaum[16] and Birsch-Hirschfeld[23]. Out of 145 cases of hereditary lues and 98 cases of acquired lues, he found but one case in each class which presented unmistakable syphilitic lesions of the stomach. The first case was in a male infant of three weeks. Autopsy showed a maculo-vesicular exanthem, pneumonia alba, gummatous cholangitis and cholecystitis and syphilitic osteochondritis. In the stomach, the mucosa over certain areas was thickened, firm, yellowish, slightly protuberant. On section through these areas the thickening was seen to embrace all the coats, the layers being fused into a homogeneous cicatricial tissue. In places there was superficial ulceration. Altogether there were five distinct lesions along the greater curvature and in the fundus. Microscopic examination showed gummatous tissue, principally in the submucosa, but extending into the mucosa and muscularis as well. The proliferation of the perivascular endothelium and the leucocytic infiltration about the gummatous areas was especially noted.

The case of acquired syphilis was in a man 23 years of age, infected two years before death. He had had frequent vomiting

for one year, severe epigastric pain after eating, for three months. Stools were diarrhœal. There was marked emaciation. At the autopsy there was found, besides the lesions in the stomach, advanced pulmonary tuberculosis and gummata of the lungs, liver and kidney, sections of which showed no tubercle bacilli. There were both tuberculous and syphilitic lesions in the small intestine. The stomach was slightly dilated: the mucosa in the pyloric region was slightly dilated: the mucosa in the pyloric region was slightly thickened and showed chronic catarrhal inflammation. In the pyloric region there was a circular ulcer, 10 cm. in diameter, situated on the posterior wall, showing a central perforation which was closed by adherent pancreas and transverse meso-colon. The submucosa about the periphery was thickened to 1 cm. and appeared on section as dense, infiltrating tissue. In other portions of the stomach also, namely, on the anterior wall, were several flat infiltrations of the mucosa and submucosa. The histological changes were practically the same as in the congenital case, and need not be cited in detail.

In addition to these two undoubted cases, Chiari found in a case of congenital lues, diffuse cellular infiltrations of the mucosa and submucosa which were probably syphilitic. In another case of acquired syphilis he found a typical peptic ulcer showing on microscopical examination no features suggesting a syphilitic origin for the lesion. In this connection, he emphasizes the fallacy of attributing all gastric symptoms occurring in syphilitics to specific disease of the stomach.

Stolper,[18] in 1896, reviewed the previous cases, accepting also those of Klebs and Weichselbaum, and gave a complete report of a personal case. This occurred in a male of 43: symptoms were pain in the stomach, loss of appetite and dyspnœa. There was a perforating ulcer of the nasal septum and other tertiary lesions. At autopsy there was found on the posterior wall of the stomach, an area of thickening with ulceration over the summit. The edges of the ulcer were infiltrated. The submucosa was replaced by firm, yellowish-gray, somewhat hyaline tissue, ¾ cm. in thickness. Microscopically, there was necrosis and cellular infiltration of the mucosa and submucosa. Plasma cells were numerous. No marked changes were found in the vessels.

In 1899, Flexner[19] recorded a case with clinical history, autopsy and histological findings. He also carefully reviewed all previously reported cases.

His case is as follows: Male, 52 years; alcoholic; symptoms, occasional vomiting, ascites, tumor and tenderness in splenic region. Later, sudden intense pain in abdomen: death. Diagnosis: Cirrhosis of liver.

Autopsy—Stomach showed a large circular ulcer (5 x 5 cm.), in fundus over greater curvature, extending to muscularis. Perforation in central portion. Edges of ulcer thick, polypoid and firm.

The liver showed syphilitic cirrhosis, with gummatous nodules. There was a large gumma in the gastro-hepatic omentum, compressing the portal vein.

Microscopically, he distinguished two stages in the syphilitic process. In the earlier stage there was a cellular infiltration of the submucosa—epithelioid cells predominating—extending into the muscularis and to a less degree into the mucosa. Here and there were scattered foci of necrosis, with fragmentation of the nuclei. The blood-vessels showed infiltration of the adventitia and obliteration in the necrotic areas.

In the later stage the submucosa is replaced by dense fibrous tissue, which runs into the muscularis. The vessels show marked obliterative changes, hyaline thrombosis, with organization. The serosa also is thickened.

Flexner concludes that the lesion is not due to a direct breaking down of diffuse gummatous tissue, but that the necrosis of the mucous membranes is brought about, secondarily, by a softening of the syphilitic tissue in the submucosa and the obstruction and obliteration of the vessels.

Aristoff,[24] in 1898, reported several congenital cases. As they show essentially the same lesions as Chiari's case and are of little clinical interest, they need not be reviewed in detail.

E. Fränkel,[25] in 1899, reported a case occurring in a male, 47 years of age, whose symptoms were epigastric pain, occasional vomiting and diarrhœa. There were found, after death, multiple abscesses of the stomach and jejunum, the submucosa, as in the other cases, showing diffuse infiltration, with epithelioid, plasma and lymphoid cells; there were marked vascular lesions, endo- and peri-arteritis, and gummatous endophlebitis.

The author believes that the lesion is the result of localized gummatous processes in the stomach wall, not necessarily originating in the submucosa. He lays considerable stress upon the share taken by the vascular lesions in producing localized necrosis.

Buday's[26] case, published in 1895, occurred in a male, 47 years of age. The clinical history, which is given in abstract, is as follows: Facial eruption for past two years. For three weeks, abdominal pain and weakness. Bloody stools two weeks ago. No vomiting or hematemesis. Examination showed large tumor in umbilical region, for which a laparotomy was performed. Death occurred on the following day.

The autopsy by Prof. von Hansemann showed the following lesions: Gummatous mesenteric lymphadenitis; gummatous ulcerations of stomach and small intestine; syphilitic ulcer of base of tongue and epiglottis; multiple gummata of liver. "In the stomach there are found near the greater curvature two almost circular ulcers, each the size of a 10-pfennig piece. One of these almost perforates the stomach wall, the other extends to the muscularis. Near the pylorus there is situated also a round ulcer about the size of a bean, whose base is raised above the surface by a small tumor * * * of the same structure as the mesenteric growths." Microscopically, the sections of the gastric ulcers showed "the typical picture of necrotic gummatous inflammation." The submucosa at the margins was infiltrated and thickened to three times the normal size. The cellular infiltration extended a short distance beyond the margin of the ulcer, but changed rather abruptly into normal submucosa. The floor of the ulcer itself is coated with a necrotic layer ½-1 mm. thick. Beneath this lies the inner layer of muscularis, the bundles of which are forced apart by "granulations" from the submucosa. The cells in the periphery of the gummatous foci were chiefly lymphoid; the central portions were formed by spindle-shaped or oval cells. The external muscular layer was uninvolved. The condition of the blood vessels is not recorded.

Additional congenital cases, confirmed by histological examination, have been reported by Bittner and Oberndorfer[27]. These need hardly be reviewed in detail.

Trinkler,[28] in 1902, in an article on visceral syphilis from a surgical viewpoint, quotes from the Russian literature, two further undoubted instances of acquired syphilis of the stomach. One of these cases (Bobrow[29]) was successfully operated upon, four-fifths of the stomach being resected. We can find no detailed description of the other case (Titrow[30]), and the original article is not accessible.

Since Trinkler's paper, we have found references to but two reported cases—those of Schmaler and Torres Barbara. We were

also unfortunately unable to secure access to either of these articles.

The number of authentic cases in the literature is therefore very small. Excluding the congenital cases of Aristoff, Chiari, Birsch-Hirschfeld, Bittner and Oberndorfer, there are but twelve which are above suspicion. A clinical analysis of so small a number is, of course, worthless, especially as the clinical data are meagre or entirely wanting in the majority of them. All the cases have been in males, the age ranging between 23 and 52 years. Epigastric pain and vomiting, both intensified by the ingestion of food, appear to have been prominent symptoms. There is no record of hematemesis—no analyses of the gastric contents.

We wish to express our thanks to Dr. George Roe Lockwood, for permission to use the clinical records, and to Dr. Charles Norris, Director of the Laboratory, for advice and assistance in the study of the material.

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FIG. 1.

Section through margin of ulcer, showing greatly thickened submucosa, in which are foci of round-celled infiltration.

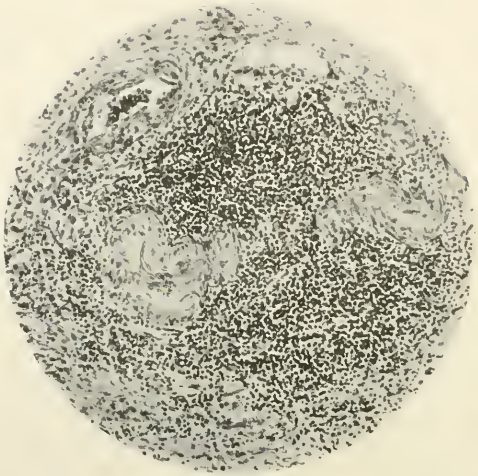

FIG. 2.

Three small blood vessels in submucosa of stomach, with extensive perivascular round-celled infiltration.

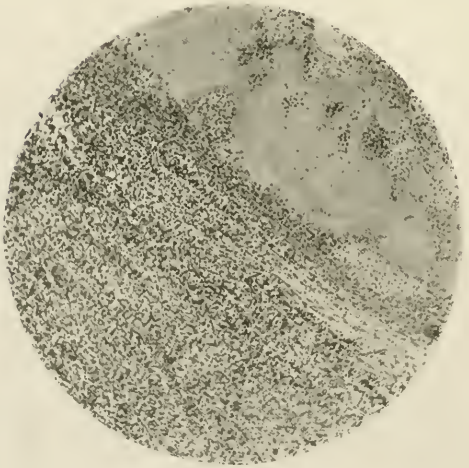

FIG. 3.

Section through wall of large eroded vessel near margin of ulcer. The coats of the blood-vessel are entirely replaced by syphilitic granulation tissue.

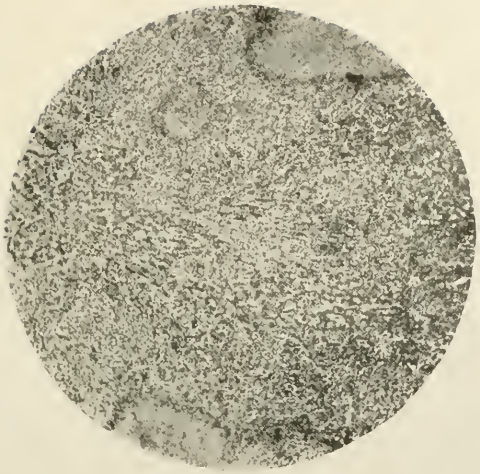

FIG. 4.

Gummata of Liver. Section shows areas of coagulation necrosis, surrounded by granulomatus tissue.

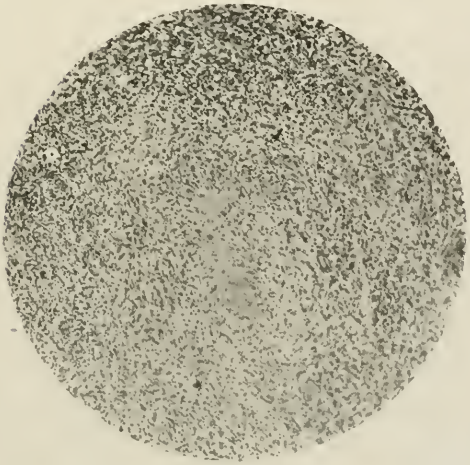

FIG. 5.

Miliary gumma of mesenteric lymph-nodes.

OBSTRUCTION OF THE COMMON BILE DUCT, WITH REPORT OF A CASE OF IRRIGATION OF THE HEPATIC-DUCT SYSTEM.*

By John F. Erdmann, M. D.,

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That all obstructions of the common bile duct eventually come to the surgeon for cure is a fallacious tenet, proven alone by numerous cases that are temporary only in their course. That all permanent and intermitting obstructions are distinctly surgical diseases, is a tenet well taken.

Some of the temporary causes of obstruction of the common duct are: Cholecystitis, with duct pressure by a distended gall-bladder; inflammatory changes in the duct or papilla, producing internal occlusive obstruction by means of infiltrate and swelling of the mucous membrane; foreign matter, as plugs of mucus, etc., and, finally, although most frequent, the passage of a stone through the duct itself.

In intermittent obstruction, we usually have a stone too large for passage through the duct, acting as a ball-valve. The symptoms appearing, then, for some reason, be it force from behind or irritation on the part of the duct, the stone is wedged or grasped in the distal portion, and the symptoms disappearing when the stone is released, either by relaxation of the irritable duct, or by some relief of the pressure from behind.

Permanent obstruction is due usually to one of two distinct processes—a stone impacted in the duct, or organic changes of benign or malignant natures. Of the latter processes, malignant changes are far more frequently found than benign stenosis.

The site of obstruction to be productive of a symptom complex sufficient to make a definite diagnosis, must be between the point of formation of the hepatic duct and the Vaterian papilla, and can be either external or internal in its relation to the duct lumen.

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Among the external pressure causes of obstruction, we have malignancy of the gall-bladder and cystic duct, the outer walls of the common duct, the pylorus, the duodenum, and pancreas, while in the internal causes we must include, with stone, benign stenoses due to former ulceration within the duct, malignancy of the Vaterian papillæ, or of the duct itself, or malignancy of the duodenum encroaching upon the orifice of the otherwise healthy Vaterian papilla.

The symptoms produced by obstruction are readily classified by the cause of the obstruction. In the passage of a stone they of necessity are acute in all phases, while in malignancy a slow onset or chronic phase is given the picture. If due to a stone, pain varying in intensity is an ever-present symptom. This is usually located by the patient himself, in the epigastrium to the right of the median line, radiating into the shoulder-blade region and even into the neck, with very frequently some girdle manifestations. Point pressure pain is elicited over the course of the duct, a point somewhat internal to that in cholecystitis.

Nausea and vomiting are often an accompanying manifestation at the onset. The temperature is usually not elevated until later complications arise, such as cholangitis, etc., although I have seen it in the onset 103 degrees, and subside in a day or two. Evidences of jaundice appear within a few hours to several days; jaundice is never a factor in cholecystitis, except as a result of pressure on the common or hepatic ducts. The stools soon lose their characteristic color and become of a light clay or putty shade. The urine presents evidences of bile, from a slight chemical trace to profound ocular manifestations. The skin, with varying degrees of jaundice, itches usually, and occasionally to such a degree as to be almost unbearable; jaundice appears first in the sclerotic of the eyeball.

The liver, while not affected at first, soon becomes engorged, and may in a few days become distinctly palpable beyond its normal relations. Digestive disturbances, chiefly referable to the stomach, arise early. Finally a distinct loss in weight is readily demonstrated.

Should the obstruction be of the intermittent variety, all the symptoms subside, either in part or entirely, only to recur when the stone is again wedged or incarcerated.

Should cholangitis arise, the clinical picture rapidly takes on a serious phase. The temperature and pulse rapidly rise, the pa-

tient has an additional facial appearance, that of an hectic flush, and a general septic evidence becomes a noted factor. The pain, instead of being limited to the duct region, spreads to the liver area, and a greater or less degree of coma is present. This latter may be present in cases where no cholangitis exists.

There should be no difficulty in arriving at a diagnosis of stone in these acute cases, except at the time of the onset of pain, as occasionally the pain is not of the characteristic variety described above, and therefore may be questioned as being that of either acute pancreatitis, gastroduodenal perforation, or a high-placed appendix undergoing an attack.

In the slow or chronic cases one can readily be led astray in the diagnosis, and great care must be taken to differentiate between chronic pancreatitis with gradual obstruction of the common duct, malignancy, or inflammation of any portion of the gastro-intestinal tract capable of producing pressure upon the common or hepatic ducts.

Should the gall-bladder be enlarged in the chronic cases, so as to be palpable, a marked suspicion of malignancy is in order. Courvoisier's law that in malignancy or external pressure to the duct, an enlarged bladder may exist, and that an obstruction by stone is usually accompanied by an atrophied or contracted bladder, holds good in a fair percentage of cases. Recently (see case cited) I had a patient, a girl of seventeen, with both obstructive cholecystitis and cholangitis, in whom the gall-bladder was enormously dilated.

Presuming that all medical aid has been given without result, or that the symptoms are of such urgency as to demand immediate surgical interference, the condition then narrows itself to a question of surgical technique and subsequent treatment.

My custom in exposing the duct is to make the incision over the right rectus, splitting the fibers in the middle and internal, or middle and external thirds, for a distance sufficiently long to explore the duct. If the stone is located or adhesions demand, the incision is then rapidly enlarged so as to give ample working room. The customary pillow under the back, depending upon the accessibility of the duct, may or may not be used. When the site occupied by the stone has been cleared of such adhesions, etc., as are necessary, and all the surrounding viscera thoroughly protected by salt solution pads, a traction suture is passed through the walls of the duct at about the upper limit of the intended in-

cision into the duct. The stone is then steadied by two fingers in the foramen of Winslow behind or below the duct, and the thumb above or in front, and an incision sufficiently long to allow of the expulsion of the stone or stones is made. After the expulsion of the stone, and proof by search that no other stones are present, a fishtail rubber tube, with from one to three openings and about ten or twelve inches long, is passed into the duct in its proximal direction, for its lower three-quarters of an inch, sufficiently to bury the apertures only. A suture of chromic catgut is then taken through both edges of the duct and includes a portion of the tube above the openings. This is tied and assures us of no slipping of the tube until the suture is absorbed. When this latter occurs, a safe way is established for further drainage, if necessary. As many more sutures are taken as may be required to close the gap made in the duct. A cigarette drain is then placed down to the site of the suture line as a precaution against leakage. The protection pads are removed after thoroughly sponging out the area with salt sponges, and the abdomen closed to the drainage site.

The removal of a stone is not always so simple a procedure. Should the stone be impacted in the lower quarter to half inch, one of two procedures can be undertaken. The transduodenal route of McBurney, or the retroduodenal method advocated and described by A. A. Berg (*Annals of Surgery,* August, 1903), and C. M. Cooper, who reported three cases successfully done by this method (*ibid,* September, 1903). Greater safety lies in the latter method, as none of the dangers of infection by intestinal contents arise that are more than possible by the McBurney transduodenal route.

It will be recalled that the tube in the duct is passed in but about three-quarters of an inch and has several perforations. These perforations allow of a certain amount of bile to pass through the distal portion of the duct in case its caliber becomes diminished so as to hug the drainage tube, while the excess of bile must perforce drain through the rubber tube. This drainage is carried off by attaching a tube sufficiently long to pass over the edge of the bed into some receptacle, usually a bottle, in which the discharge can be seen and measured. When the physical character of the bile begins to assume a normal aspect, I am in the habit of pinching the tube an hour out of every eight for the first day, then two hours at a time, etc., so that in a short time, at the

end of the fourth or fifth day of apparent normal bile, the tube, if still in, is pinched or shut off for about twelve hours out of twenty-four. I deem this an essential procedure, as the system absolutely demands this great secretion for the proper metabolism of the food intake.

It has been, and is yet, a custom of mine, during the first few days, to give oxgall in doses of five to ten grains every four hours, and I feel that noted benefit is derived in those cases where the quantity of drain seems excessive. After the drainage tube comes out (from ten to twelve days) the skin of the abdomen requires, in most cases, constant attention to prevent a most pernicious dermatitis. This is readily accomplished by the use of boric or ichthyol ointment, and cleanliness by frequent dressings. The fistula usually closes in from two to three weeks.

In cases of malignancy and stricture, should the growth or stenosis be distal to the cystic duct and non-removable, then a cholecystenterostomy is in order, while if it is removable, excision with end-to-end, or end-to-duodenum, etc., anastomosis should be done. If it be proximal to the cystic duct, operative procedure becomes almost impossible.

The history of the case referred to above is of such interest from the viewpoint of the age of the patient, the enormous dilation of the common duct, and the extent of the operative procedure, that I include it as a most interesting conclusion to this paper.

A girl, seventeen years of age, of Russian-Jewish parentage; no history of typhoid fever; always constipated; perfectly well previous to her landing in America two and a half years ago; a victim of favus; entered Gouverneur Hospital on April 1 of this year, and gave the following history: She had been jaundiced for four weeks. Has had occasional cramp-like pains in the upper abdomen on the right side. On the day before admission she had an attack so severe in its character and duration as to necessitate urgent attention.

This additional history was obtained subsequent to the operation: On coming to America two and one-half years ago, she was ill for six weeks; had amenorrhea and pains similar to the present; no jaundice noticed. About two years before admission she was jaundiced for about one year. During this time she had a dull, aching pain in the right hypochondrium, which was occasionally intensified into an acute stabbing pain in the same region.

This was followed each time by dark blood in the fecal discharges. She does not remember the color of the stools or urine. No gallstones were noticed in the stools. During this time she says she vomited about three times a week, and each time the vomiting was followed by relief. Had to leave her work at times and remain in bed on account of the pain; would feel warm and cold, and drink much water. Has been more or less jaundiced ever since that time, and has never been entirely well. These attacks of jaundice were accompanied by profound itching of the skin. She says the pain in the back, directly behind the gall-bladder, was more severe than in front. All this time she has had severe headaches and indigestion, "yellow vision," and black spots before the eyes.

Upon admission the patient was delirious and in coma. Well nourished, deeply jaundiced skin and conjunctivæ. Scalp, favus over the greater portion. Chest, negative. Abdomen, tenderness and rigidity over the gall-bladder region; also markedly tender spot about one inch above the umbilicus and somewhat to the right. Liver dullness four inches below the costal arch and extending downward over what appeared to be an enlarged gall-bladder. Pulse, 106 to 120; temperature, 103.4 degrees by mouth. The temperature on April 2 fell to 99.5 degrees, pulse 88. On the following day the temperature was 104.6 degrees, pulse 152; delirium and coma were present, and the patient looked septic.

Operation was done within forty-eight hours of admission and in the third day of the attack. Upon exposing the liver, it was found to be nodulated and congested to a very deep degree, exceptionally hard to the touch, and greatly enlarged. The gall-bladder was about eight inches long and three inches in diameter at its fundus; tense, and buried in an omental mass, highly discolored and presenting spots of color through its serous coat that were considered (and so proven after opening) gangrene of the mucous coat.

After liberating the bladder from its omental encapsulation, a stone was palpated in the cystic duct. By following the duct to the common, a mass of stony hardness, over three inches in length and larger than an ordinary thumb, occupied the site of the common duct. Cholecystectomy, choledochotomy, etc., were decided upon. While releasing the gall-bladder from its liver sulcus, it ruptured, expelling fully ten ounces of the most foul smelling and appearing contents that I have ever met with. The

bladder was then rapidly excised, and a stone about three-quarters of an inch long by a half inch in its largest diameter was removed from the cystic duct. Two pairs of forceps grasped the sides of the cystic duct, which was then split to the common duct This latter and the hepatic were then split for a distance of an inch and a half. Two stones, seven-eighths of an inch in diameter and measuring two inches long, were expelled, while a third one was located in the pancreatic and retroduodenal portion of the duct. This was also seven-eighths of an inch in diameter and a fraction over an inch long. This latter stone was so thoroughly fixed that great difficulty in its expulsion was met with. Owing to edema, adhesions, etc., neither the transduodenal nor the retroduodenal operations could be done. Having a large duct, and a large opening in it, I was able to fragment the stone with a small instrument introduced into the duct, part of the stone being pushed through the papillary opening, and the remainder delivered by scoop through the choledochotomy wound.

Examination of the hepatic ducts was then made with the little finger. These were blocked with stone and biliary detritus. Upon removing these materials there was a profuse gush of the same foul-smelling and appearing material as was present in the gall-bladder.

Re-examination of these ducts proved the presence of more of this same material. A blunt curette was then passed into both right and left ducts, bringing out quite a quantity of detritus, and then, although I had never seen nor heard of irrigating the liver ducts, I introduced a quarter-inch irrigating tube and flushed the liver with saline solution through these ducts. During this flushing process one could readily see the liver, which had subsided considerably as a result of the emptying of the ducts, rise and fall with the entrance and discharge of the solution.

A tube was sutured into the duct, several sutures taken to close the balance of the incision in the duct, and then a deep incision was made into the liver substance of the superior surface of the right lobe, for about two and a half inches in length. This was done as an extra means of drainage, and was accompanied by a fair hemorrhage, but was readily controlled by gauze. The exposed area was sponged with salt solution, and the wound closed to the drain site. Time occupied, one hour and ten minutes.

The temperature dropped to 100 degrees by rectum, and remained on a line between 99.4 degrees and 100.4 degrees for six

days, then became normal and remained so except for an occasional constipation rise of one degree. Fifteen ounces of dirty, foul-smelling bile were collected the first fifty hours.

April 7—Patient doing nicely; bile clearing up.

April 11—Stools, which were clay color before operation, still so. Tube came away; patient passed fragments of stone by the bowel, that had been forced through the papillary opening.

April 17—Stone size of pea discharged through the wound. This must have come from the liver. Stool well formed and yellow. Sinus in abdominal wall doing well.

April 22—Patient sat up for the first time, that is, on the nineteenth day after the operation.

May 5—Patient was discharged at her own request, thirty-three days after the operation.

The urine varied in specific gravity, from 1030 to 1016; it was acid except on April 5 and May 4, when it was found to be alkaline. Some blood was found on April 4. The blood count on April 3 showed white blood corpuscles, 20,000; polynuclear differential, 81 per cent.

A CASE OF PRIMARY CARCINOMA OF THE BILE DUCTS.

By A. M. Pappenheimer, M. D., *Pathologist.*

(*From the Pathological Laboratory of Bellevue Hospital.*)

Dr. A. M. Pappenheimer presented briefly a case of primary carcinoma of the common bile duct, which showed some rather interesting features. He was indebted to Dr. Nammack for permission to use the clinical records.

The patient was a butcher, fifty-three years of age, who was admitted to the Fourth Medical Division at Bellevue Hospital, on October 20, 1906. He was said to have been an excessive drinker of beer and whiskey. The symptoms for which he was admitted dated back three months, when he first noticed that he had become jaundiced; shortly afterward, his abdomen began to swell. The icterus deepened gradually in intensity, and his abdominal distention became more marked. At the same time his appetite failed, and he began to lose weight and strength. He had no severe abdominal pain at any time and suffered only from a sensation of heaviness and distention in his abdomen. His bowels were constipated until within three days of admission, when he had a sharp attack of diarrhea. Following this there was complete obstipation, unrelieved after his entrance into the hospital by cathartics or enemata. There had been no vomiting or bloody stools.

Upon admission the patient was very weak, prostrated, somewhat emaciated, and slightly delirious. There was marked icterus of the skin, scleræ and mucous membranes. The tongue was dirty and heavily furred; the breath foul. The heart was displaced upward; and the sounds were feeble and irregular. The pulse was small, weak and arrhythmic. The lungs showed general sibilant and moist rales, with dullness and diminished respiratory murmur at both bases. The abdomen was much enlarged and distended. There was a diffuse fullness in the epigastric region, a shallow depression across the umbilicus, and, again,

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a diffuse prominence in the lower portion of the abdomen. Percussion showed general tympany. There was rather marked general tenderness on palpation. The abdominal wall was tense, but there was no muscular spasm. None of the viscera were palpable, and no tumor masses could be distinguished. Liver dullness began at the fourth rib in the right mammary line, and extended not quite to the free border. The spleen did not appear to be large on percussion, and could not be felt.

The urine showed a trace of albumin, occasional hyalin and granular casts, and bile in large amount. The blood showed a leucocytosis of 28,000, and a slight secondary anemia.

The patient lived three days after admission, dying with symptoms of cholemia. He became, first, very delirious, then lethargic and comatose. The abdominal distention increased rapidly, and as his bowels remained obstinately constipated, a suspicion of intestinal obstruction was entertained. His condition, however, did not justify an exploratory operation.

At the necropsy, performed six hours after death, there was found intense light yellow icterus of the skin and conjunctivæ. All the body tissues and fluids were bile stained. The abdomen was prominent and tense; the panniculus scant. The peritoneal cavity contained several liters of turbid, dark yellow or brownish fluid, in which floated large yellow fibrin flocculi. The omentum was rolled up over the surface of the liver, and was adherent between the anterior surface and the diaphragm. The intestines were much distended with gas, displacing the diaphragm upwards. The peritoneum in a few places showed slight recent fibrinous deposits. The liver extended not quite to the costal margin. The heart and lungs presented no noteworthy lesions. The liver was slightly reduced in size, and greenish in color; and the surface was finely granular. The organ was very firm, and on section showed a fine, irregular cirrhosis. The large intrahepatic bile ducts appeared to be dilated, and were filled with dark green bile under considerable pressure. The gall bladder was much distended, projecting about three fingers' breadth below the liver, and lying with its long axis almost parallel to the lower border. The wall of the gall bladder appeared slightly thickened; but there were no adhesions. Upon opening the duodenum, thick, greenish bile escaped from the papilla, without pressure upon the bladder. Beneath the mucous membrane, the transduodenal portion of the common duct could be seen and felt as a rigid, thick-walled cylinder.

After hardening, a series of transverse sections was made through this portion of the duct, including the overlying duodenal wall and the adjacent pancreatic tissue. It was then found that this portion of the common duct for a distance of several centimeters proximal to the ampulla showed a diffuse and uniform thickening of the wall. The tissue of the duct was grayish white and very firm. There was no infiltration of the duodenal wall, and only in one section did the new tissues appear to encroach slightly upon the head of the pancreas. The wall of the duct measured about 5 to 7 mm. in thickness. The lumen was slightly narrowed, but was nowhere completely stenotic. A small probe could be readily passed up through the papilla. The ampulla itself appeared somewhat rigid and thickened, the mucous membrane normal. Wirsung's duct was found to open into the ampulla and was patent.

The common duct proximal to the thickened portion, as well as the cystic and hepatic ducts, was dilated to the size of one's thumb. At one point, about 2 cm. below the junction of the cystic and hepatic ducts, the common duct showed slight thickening of the wall. The overlying mucosa was normal. The periportal and peripancreatic lymph glands were moderately enlarged, but on section showed no evidence of malignant involvement.

The remainder of the organs showed no features of special interest. No secondary malignant deposits were found in any of the viscera. The small intestine contained colorless, rather greasy, fecal matter; but in the large intestine the stools were bile stained, showing that complete biliary obstruction had not occurred until shortly before death. To explain the acholic feces in the small intestine, in view of the fact that the duct at autopsy was patent throughout we may assume a muscular spasm in the infiltrated portion, as has been suggested by Rolleston, or possibly an interference with the normal peristalsis along the ducts, or a transient plugging of the orifice of the ampulla with mucus or inspissated bile. It is very likely that atony of the gall bladder from over distention may also have been a factor.

Cultures made from the gall bladder on ascitic agar proved sterile. On opening the bladder the mucous membrane was found to be normal; there were no calculi.

Histologically, the thickening of the duct proved to be due to an infiltrating scirrhous carcinoma of the columnar celled type, undoubtedly taking its origin from the mucous membrane of the

duct. The acinal structure was fairly well retained, even to a considerable depth in the wall; but lying in the lymph spaces of the stroma were solid nests or islands of closely packed cells, in which the columnar form was no longer discernible. The cells which composed these islands showed some very remarkable features. Many of them were exceedingly large. Frequently the cells, crowded together, had lost their individual outlines, and formed large syncytial protoplasmic masses containing a number of swollen vesicular nuclei. There were also some curious structures within these cell nests. In the midst of a group of cells there was frequently to be found a sharply outlined circular or spherical space, crowding the adjoining epithelial cells with their contained nuclein into crescentic shape. Within this circle lay a single epithelial cell, showing always more or less degenerative change. At a later stage of the same process there was a very large but clearly circumscribed vacuole in which could usually be found the degenerated but structureless remains of an epithelial cell.

The tumor was found, in sections from one block, to infiltrate the pancreas for a short distance, penetrating the lobules as well as growing into the interlobular connective tissue. The pancreatic ducts were not dilated, and were quite normal in appearance, proving that Wirsung's duct was not obstructed by the growth. In confirmation of this there was found in a section through the ampulla itself only an adenomatous hyperplasia and no carcinomatous change.

The liver showed an advanced cirrhosis of irregular type, with a rather cellular connective tissue. There was considerable stasis of bile in the bile capillaries, and in places, bile pigment within the liver cells. On the other hand, the bile ducts in the portal spaces did not appear in the sections to be greatly dilated. A few small areas of necrosis were found, in what portion of the lobule was not clear. There was some evidence of acute inflammatory change, as polynuclear leucocytes were found throughout the section between the liver cells and in the periportal spaces.

A CASE OF MULTIPLE PRIMARY NEOPLASM OF THE SMALL INTESTINE.

By Alwin M. Pappenheimer, M.D., *Pathologist.*

(From the Pathological Laboratory of Bellevue Hospital.)

The following case, occurring in the service of Dr. Cyrus J. Strong, is of interest chiefly from a pathological point of view.

Clinical data are almost lacking. The patient was admitted in a delirious and almost moribund condition. From relatives it was learned that he had been ill but four weeks and had worked up to within two days of entrance to the hospital. His symptoms during this period were cough and "pains in the back." No history of any abdominal symptoms could be elicited.

The patient, for a number of years, had been excessively alcoholic. Strong family predisposition to tuberculosis was ascertained, several brothers having died from this disease.

The physical examination on admission showed moderate cachexia and the signs of an advanced tuberculous lesion in the right lung. The patient became rapidly worse, and died three days after admission.

The following is a transcript of the post-mortem findings:

F. R., age 38; autopsy on June 19, 1903, 21 hours after death. Body of a well-developed male; marked rigor mortis; muscles dark red; panniculus ¾ inch over abdomen; diaphragm 4th right space, 5th left; appendix normal; mesentery contains a large amount of fat; omentum free; no fluid in peritoneal cavity.

Thorax—Left pleural cavity: Old fibrous adhesions over upper portion of lower lobe posteriorily. Apex free—no fluid.

Right pleural cavity: Firm adhesions over upper and lower lobes. Parietal pleura is studded with miliary tubercles over costal, vertebral and diaphragmatic surfaces. There is considerable œdematous fibrin over the right lower lobe. On the pleura of the upper lobe, numerous miliary tubercles and scars, and old calcareous nodules.

On the right pleural surface of the diaphragm are several flat, circular or oval tumor (?) masses, about ½ cm. in height and from 1 to 5 cm. in diameter; section of these shows firm gray-

ish tissue, which is vascular and yields no juice on scraping. A similar flat nodule is situated on the mediastinal pleura over the vertebræ. There are also very numerous miliary tubercles (?) on the diaphragm and costal pleura.

Left lung, on section, shows marked general congestion and œdema. Bronchi are congested; lymph nodes at the hilus are anthracotic and small; right lung, on section, upper lobe, and upper portion of the lower lobe, is the seat of numerous disseminated miliary tubercles. There are also a large number of firm, whitish and dry areas, many of them encapsulated, averaging from ½ to 1 cm. in size. In the upper lobe are a number of irregular, ragged, thin-walled cavities, apparently not containing pus.

Parenchyma of the right lung is congested and œdematous in the dependent portions. Bronchi are congested. Bronchial lymph nodes anthracotic and the seat of miliary tubercles.

Heart—Pericardial sac contains slight excess of clear fluid. Heart is of normal size. Valves and orifices normal, save that there is slight thickening at the edges of the mitral segments. Cavities contain fluid blood. Foramen ovale closed. Heart muscle is pale, somewhat friable. Coronary arteries are tortuous, but show no atheroma. The aorta, thoracic and abdominal, is normal.

Liver—Weighs 3 lbs., 14 oz. Edges rounded, extending about a finger's breadth below costal arch in right mammary line. Color of surface is yellow, mottled by irregularly sized, depressed reddish areas. There are seen on the superior surface several small, flat whitish nodules; section of liver is firm. Liver tissue is brownish-yellow in color, with irregular, depressed reddish areas. There are seen also a number of spherical whitish nodules, varying in size from 3 or 4 to 15 mm. in diameter, scattered throughout the substance.

Gall-bladder contains yellowish bile and a few irregular concretions. Mucosa normal.

Spleen—Weighs 8 oz. Capsule is wrinkled. Consistency is firm. On section, there is marked congestion, and increase in interstitial tissue.

Pancreas—Pale; shows post-mortem change.

Stomach—Normal.

Small Intestine—The intestine, for a distance of about three feet above the ileo-cœcal junction, is the seat of a number of

scattered submucous nodules. These are from 3 to 10 mm. in diameter, roughly spherical in shape, firm, grayish-white and not caseous on section. They are circumscribed, but not encapsulated, and do not infiltrate the adjacent tissue.

The mucous membrane over the smaller nodules is intact, the nodule projecting as a small, rounded prominence, into the lumen of the gut. Over others, the mucous membrane is partly or entirely ulcerated. The largest ulcers are irregularly oval, with the long axis lying transverse to the gut. In these the nodular growth is situated at one end of the ulcer. The mucous membrane is drawn over the nodule on the healthy side, but deficient on the ulcerated surface. The margins of the ulcers are raised and infiltrated, the base covered by a thin slough.

In the floor of one of these ulcers, and visible from the peritoneal surface, are seen several smaller, flat, whitish, tubercle-like nodules. The ulcer which is situated highest above the ileocoecal junction shows no nodular growth, but closely resembles a typical advanced tuberculous ulcer; it is oval, transverse to the long axis of the gut, situated opposite the mesenteric attachment; the edges are raised, indurated and overhanging. No tubercles are seen in the floor, however, and the serosa is intact.

The small intestine shows no other lesions, save slight injection of the smaller vessels of the valvulæ conniventes. There is no appreciable change, either dilatation or stenosis, in the lumen of the gut at the site of the lesions.

Large Intestine—Shows moderate hyperæmia. There are no nodules or ulcers.

Mesenteric and Retro-Peritoneal Lymph-Nodes—The mesenteric lymph-nodes are all slightly enlarged. There is found one which on section is very firm, pink in color, with whitish periphery. Similar glands are found situated in the retroperitoneal tissue below the hilus of the left kidney.

Adrenals—Normal.

Kidneys—Weigh together, 15 oz. Capsule is adherent, leaving, however, a smooth surface. Cortex averages normal width. Markings very indistinct. Color pale. Vessels injected. At the base of one pyramid of the left kidney is a small, whitish tubercle-like nodule.

Pelves and ureters normal.

Prostate—Large and fibrous, and on section shows numerous brownish, gritty granules.

Histological Examination.

Section Through Intestinal Nodule—Medium-sized ulcerated nodule. The mucosa adjacent to the nodule shows a denudation of the epithelium over the crests of the villi. The glandular epithelium in the crypts is intact and shows no degenerative changes. Over the summit of the nodule the mucosa is entirely wanting, the floor of the ulcer being formed by a thin layer of necrotic tissue in which are seen fragmented nuclei, small, round cells, and large bacilli in clumps. The tufts of the villi show inflammatory infiltration with small, round cells.

The submucosa is thickened by a nodular growth of new tissue, which shades gradually into the normal tissue on either side of the ulcer. Under high magnification this is seen to be made up of a highly cellular tissue through which course many thin-walled capillary vessels. The cellular elements vary greatly in size and appearance and show lawless arrangement which at once suggests an actively growing tumor. There can be distinguished (1) large spindle cells, with oblong or oval vesicular nuclei, showing a distinct chromatin network and frequently a large nucleolus; (2) polyhedral cells, with large nuclei, similar to the foregoing; (3) many of these show karyokinetic figures; in others the nuclear substance is fragmented into irregularly disposed, intensely staining chromatin masses; the cytoplasm of these mitotic cells stains deeply with eosin; (4) large, polyhedral cells, containing three or four very large vesicular nuclei (giant cells); (5) large cells, containing a single deeply-stained homogeneous nucleus at the periphery, the cytoplasm taking the eosin rather intensely; (6) a few cells showing coarse, reddish granulations with polychrome-methylene blue, but having a small, circular nucleus (mastzellen).

In addition to these larger cells there are seen numerous small round cells of the usual lymphoid type. These are scattered diffusely amongst the other cells, or, as is seen, particularly near the surface of the ulcer and at its margin, aggregated into dense groups.

With Van Gieson's connective tissue stain no definite reticulum can be made out, although there are irregular strands of connective tissue fibrils, taking a deep, reddish color. There is no limiting capsule or alveolar structure. Towards the periphery of the nodule, in some of the sections, are seen a few fairly typical miliary tubercles. These show a central necrotic area, in which

are a few pycnotic nuclei and one or more large giant cells—large, homogeneous bodies with ten or twenty deeply staining, radially arranged nuclei. The nuclei show fine tapering processes, pointing centrally. The necrotic areas, at least most of them, are surrounded by a moderately dense aggregation of small lymphoid cells; some, however, lie in the midst of the tumor tissue.*

Section Through Small Submucous Nodule—The mucous membrane and muscularis mucosæ overlying the nodule are intact and show no pathological change. The nodule consists of an aggregation of cells, presenting the same diversity of size and form as the larger growths. It lies entirely in the submucosa, and though there is no trace of a limiting capsule, the nodule is fairly circumscribed and does not infiltrate the muscular layers. The section shows conclusively the origin of the new tissue in the submucosa.

Section Through One of the Retroperitoneal Lymph-Nodes—There is no trace of normal lymphoid tissue. The prevailing type of cell is large and fusiform or spindle-shaped with tapering processes. The nuclei of these cells are oval or cylindrical in shape, lying in the long axis of the cell. They exhibit a deeply-staining chromatin network and occasionally a distinct nucleolus. The cytoplasm stains deeply with eosin. The tapering ends of the cells in places show slight longitudinal striation. Cross-sections of these cells through the nucleus show the latter to occupy the whole diameter of the cell, in this respect resembling smooth muscle fibres.

There are seen also polyhedral cells, having a number of tapering fibrillary processes, and small giant-cells, containing several vesicular nuclei, usually occupying the central portion of the cell.

Karyokinetic figures, most of them atypical, are very numerous. In many cells are seen swollen nuclei, with transverse lines of fission, apparently about to undergo amitotic division.

The arrangement of the spindle cells is highly irregular, although in places there are loose strands or fasciculi in which the cells lie in the same axis. These strands of slender spindle cells resemble, somewhat, bundles of smooth muscle fibres, although the individual cells lie loosely separated in a fibrous stroma.

There are a few scattered areas of necrosis which consists apparently of degenerated tumor tissue, in which lie a few pycnotic

* No tubercle bacilli could be demonstrated in these caseous areas after careful search.

nuclei. These areas do not in the least suggest tuberculous caseation. There are no giant cells of the Langhans type.

Throughout the gland are very numerous, circular vacuoles, which appear to be large fat cells.

There is a moderate number of thin-walled capillaries whose endothelium is much swollen.

Section Through One of the Mesenteric Lymph-Nodes exhibits the same general histological structure. The necrotic areas, however, are more numerous, and the cells show a greater diversity of form. Again, there is no rest of normal lymphoid tissue, and nothing suggesting tuberculous involvement.

Section Through the Diaphragmatic Tumor Mass—The tissue is very cellular. There are numerous large, polyhedral or spindle-shaped cells, with vesicular nuclei. Karyokinetic figures are even more abundant than in the intestinal nodule. There are a few strands of spindle-cells, which suggest smooth muscle fibres. No striated fibres are seen in the section.

The cells lie in a loose fibrillated reticulum, staining red with Van Gieson's stain.

The tissue is highly vascular; there are many new-formed capillaries whose endothelium is swollen.

There are irregular areas of necrosis, staining diffusely with eosin, but showing no giant cells or tubercle formation.

Liver—The parenchymal cells in places show marked fatty infiltration; in others, granular degeneration. The reddish areas noted macroscopically correspond to irregularly distributed areas of venous congestion; there are also small areas of extravasation, especially in the vicinity of the tumor nodules. Throughout the section there is a slight degree of interlobular cirrhosis, which is irregular in distribution.

The whitish nodule is composed of tumor cells, whose arrangement and general appearance closely resembles that seen in the intestinal lesions and diaphragm. Spindle cells which might be interpreted as smooth muscle fibres are not numerous, polymorphic cells in all stages of active mitosis predominating. The tumor tissue is less vascular than the diaphragmatic nodule.

There is no trace of a limiting capsule. The liver cells adjacent to the tumor are compressed by the growth.

Lungs—Section shows typical lesions of chronic miliary tuberculosis. There are disseminated areas of coagulation-necrosis

and miliary tubercles with giant cells. There is marked interstitial fibrosis and general congestion, some of the alveoli containing extravasated blood.

No metastatic tumor nodules seen on section.

Spleen—Congestion and hypertrophy of trabeculæ; no metastases.

Kidneys—Show congestion and moderate granular degeneration of the epithelium of the convoluted tubules.

Sections taken from the *testes*, *pancreas*, *adrenals* and *heart muscle* show no noteworthy lesions.

Unfortunately, we were unable to examine the marrow of the long bones.

Using the term "sarcoma" in a broad sense, it seems to us that the case is one of multiple primary sarcomata of the small intestine, with metastases in the mesenteric and retroperitoneal lymph-nodes, liver and diaphragm. An alternative possibility, which must be considered, is that we are dealing with an unusual form of intestinal pseudo-leukæmia.

The tumors described in the literature as primary sarcomatous growths of the small intestine differ widely in their gross appearance and histological structure. Some are diffuse infiltrating growths, such as an extreme instance described by Lehman,[1] in which the entire intestine from rectum to jejunum was converted into a rigid, thick-walled tube; others are large, localized tumors, which may or may not grow into the mesentery and adjacent organs. Less frequently, as in our case, there are a number of discreet primary foci. The origin of the growth from the submucosa can be demonstrated in almost every instance. In but one of the articles accessible to me do the authors, Babes and Nanu,[2] refer the origin of the tumor, described as myosarcoma, to the muscularis.

Metastases may or may not be present. When they occur, the peritoneum, regional lymph-nodes, kidneys, liver, spleen, diaphragm and pleura—are oftenest involved.

The lumen of the gut may be unaltered, if the growths are small. More frequently, there is a cylindrical dilatation at the site of the tumor; this may be so extreme as to form an aneurysmal sac of large dimensions. Again, there may be stenosis caused by invagination (Wallenberg[3]), or by adherence and kinking of the intestinal coils (Pick[4]). Complete obstruction has been caused by a sarcoma occurring in an inguinal hernia (Walden-

strom and Akerberg[5]). In a much-quoted case (Stern[6]), a child dying four days after birth, was found to have a congenital lympho-sarcoma of the ileum.

Widely as these cases differ, I have been able to find but two cases which closely resemble in the gross the one here described. These are the multiple growths of the ileum, described and pictured by Schlagenhaufer[7]. No detailed histological description is appended, but we are told that the tumors proved to be "alveolar sarcomata" and "endotheliomata." Two cases both occurring in tuberculous patients presented somewhat similar lesions, although there were no metastases in the other viscera. The first (Jardet[8]), in addition to an advanced pulmonary tuberculosis, showed numerous transverse ulcers of the ileum, averaging about 2 to 4 cm. in size. The edges of these were infiltrated, and the bases showed hard, grayish plaques, visible on the peritoneal surface. Histological examination showed "a lymphadenoma." The second case (Nothnagel[9]) was a lympho-sarcoma, in which the sarcomatous nodules arose on the site of healed tuberculous ulcers. The autopsy by Prof. Weichselbaum showed that there were "throughout the small intestine numerous cicatrices of tuberculous ulcers * * * also tubercular ulcers, annular or partly encircling the gut. * * * In addition, in the great majority of the cicatrices, nodular grayish white masses were seen. These resembled little tumors, and, on microscopic examination, were found to be lympho-sarcomata. Size, ½ to several mm. in diameter. In some of the cicatrices little nodules were found, either at the margin of the ulcer or at the base; in other cicatrices the margin was formed by a continuous tumor mass. * * * Only one nodule was found not related to definite cicatrix."

As regards the gross appearance of the lesions, Nothnagel's case agrees rather closely with ours. Both cases illustrate the coexistence of tuberculosis with malignant processes—an association so frequently encountered with the lympho-sarcomata and the various leukæmic and pseudo-leukæmic processes. In Nothnagel's case the tuberculous process very evidently antedated the malignant, the neoplasm being engrafted upon cicatrizing ulcers. In our case the reverse would seem the more probable. The few miliary tubercles seen in some of the ulcers are extremely recent; and the smaller not ulcerated nodules show no features which might be construed as tuberculous inflammation.

As to the histological structure of the intestinal sarcomata, there is even less uniformity than in their gross characters. Because of this morphological diversity, and because also of the lack of a consistent and definite nomenclature, a great variety of sarcomatous tumors have been reported, whose exact classification is not easy to decide.

The group most often encountered comprises those growths taking their origin from the lymphatic structures of the intestine. Leaving aside the unsettled question of the relation of these tumors to the true sarcomata, we may place under this heading the "lymphosarcomata," "lymphadenomata," "malignant lymphomata," and possibly also the tumors described by Lehman[1] as "endothelioma interfasciculare" and by Oberndorfer[10] as "Lymph-gefäss Carcinome." All these forms of intestinal neoplasm have this structural feature in common, that they consist of a fibrous reticulum supporting cells more or less closely resembling the normal lymphoid elements.

A second group may be taken to include the round and spindle-celled sarcomata. These are distinguished from the first group, less by a difference in the appearance of the cellular elements than by the absence of a definite fibrous reticulum. Although we encounter here a wide variation in the size and shape of the tumor cells, there is less confusion in nomenclature and a closer rapprochement to the types of sarcoma as it occurs elsewhere in the body.

There have been described, finally, melanotic sarcomata and leio-myosarcomata. The former variety, though occurring not so infrequently in the rectum, is a pathological rarity as a tumor of the small intestine. Myosarcomata are scarcely more frequent (Wallenstrom and Akerberg; Babes and Nanu).

The absence of a definite reticular structure and the great diversity of the cell-types—especially the predominance of epithelioid and giant-cells—seem fair reasons for not classing this tumor with the lympho-sarcomata, unless indeed we believe the cells to be derived from the endothelial lining of the reticular spaces. We were unable to demonstrate such an origin for the tumor cells, nor can it be proved that the nodules start in the lymphatic tissue. In sections through the smallest, and presumably the most recent of the intestinal nodules, no lymphatic tissue can be seen.

If we exclude the class of lympho-sarcomata, we are forced to describe the growth as a "mixed-cell" sarcoma, presenting, I

believe, a different histological picture from the hitherto described forms of intestinal sarcoma. There remains only the possibility that we are dealing with a mixed growth—a leio-myosarcomata —and this is rendered remote by the fact that the tumors take their origin from the submucosa.

As an interesting alternative to the conception of these tumors as malignant growths in the stricter sense of the term is the possibility that the lesion is a primary pseudo-leukæmia of the intestine, and that the nodules in the liver, diaphragm and lymph-nodes are not true metastases, but part of the same general process. Cases of pseudo-leukæmia without involvement of the peripheral glands, have been reported by Fisher,[11] Longcope,[12] Simmons[13] and others. Histologically, the masses of epithelioid cells with vesicular nuclei, the multinuclear giant cells, and the other structural features of the new tissue, give a picture not unlike that considered by Reed,[14] Longcope and Simmons so distinctive of Hodgkins disease. If the diagnosis is to be based upon morphology, one is strongly tempted to group our tumor with this class of adenopathies.

Without attempting to enter fully into the question, certain objections to this interpretation of the lesions may be mentioned. The origin of the growths from lymphatic tissue cannot be proven. The spleen is uninvolved, showing neither a diffuse enlargement, nor the presence of localized lymphomatous masses of characteristic structure. The collections of eosinophile cells so frequently met with in pseudo-leukæmia, and considered by Reed of diagnostic significance, are missing. Whereas, in pseudo-leukæmia the process begins with an abnormal proliferation of normal lymphoid tissue, showing in its earliest phase as an aggregation of small round cells, in this case the newest growths exhibit the same marked diversity of cell form as the older lesions. It seems to me that these are valid objections against this interesting but untenable view.

In our ignorance of the etiology and relationships of these various forms of intestinal neoplasms it would seem to be largely a matter of individual experience what descriptive terms we apply to this case. It has seemed to use that the term " mixed-cell " sarcoma is the most applicable of those at our disposal, and the case is of interest chiefly as illustrating the difficulty in arriving at an exact pathological diagnosis.

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ENTERIC AND MESENTERIC CYSTS, WITH REPORT OF AN UNUSUAL CASE.*

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Cystic tumors occupying the lumen of the intestinal canal are of such rare occurrence as to make them of pathological interest rather than of surgical importance; yet the possible origin of certain of these cysts would seem to classify them with other tumors similar in character, but different in location, which, from their comparative frequency, render them of practical importance to the surgeon.

Rokitansky[5a] says that even in the vast amount of pathological material he has seen at the Vienna General Hospital cysts of the intestinal wall are great rarities.

After a fairly careful search in the literature of intestinal tumors I have been able to find a description of but one case at all resembling my own, given below, and that only in certain particulars.

History of Patient and Description of the Operation and of the Cyst—January 7, 1905; Bellevue Hospital (service of Dr. Chetwood). J. K., male, aged twenty-three years; Japanese. Nineteen years ago he had an unidentified eruptive fever. Bowels have always been regular, excepting during four attacks of his present trouble, to be described later. Seven years ago, without any apparent cause, he had an acute attack of severe pain in the right lower quadrant of the abdomen. He was confined to bed with this attack for two months, during which time he had several attacks of vomiting, intermittent constipation, and some fever. He was treated by the application of an ice-bag, locally, and by internal medication. His convalescence was gradual.

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He had no recurrence of these abdominal symptoms, with the exception of infrequent pains in the right iliac fossa, until two years ago, when he was suddenly seized with severe pain in this locality, accompanied by fever. He did not go to bed and recovered at the end of two weeks.

With the exception of infrequent attacks of slight pain, he remained free from all symptoms referable to his abdominal condition until thirteen days ago, when he was suddenly seized with a severe pain in the right iliac fossa, which compelled him to take to his bed. He did not vomit, but thinks he had some fever. The next day he was brought to Bellevue Hospital, where he was treated by the application of an ice-bag to the abdomen. His temperature, while in the hospital, remained low, his pulse ranged between 90 and 100, and he had a leukocytosis of about 12,000. At the end of five days he was discharged, cured.

Two days ago he had an attack of pain similar to his previous ones. He kept at work for twenty-four hours, when the pain became so severe that he was compelled to go to bed, and had two attacks of vomiting. He then returned to Bellevue Hospital and soon after his admission had four attacks of vomiting.

Present Condition—His temperature ranged between 97.5 degrees and 99.5 degrees; his pulse between 100 and 116; respirations between 20 and 28. The blood count showed 4,000,000 red cells; 15,000 white cells. The differential count gave the following result: Polymorphonuclear cells, 85 per cent.; large lymphocytes, 8 per cent.; small lymphocytes, 7 per cent. The bowels were obstinately constipated after one small passage, resulting from an enema. Examination showed slight tenderness and resistance at McBurney's point, but no tumor could be made out. A diagnosis of recurrent catarrhal appendicitis was made and the patient was prepared for operation.

Operation—The cæcum was exposed through the usual intermuscular incision, and was drawn into the wound. The appendix was inspected and found to be normal in every respect. It lay in the retrocæcal position. Further inspection disclosed the fact that the lumen of the cæcum was occupied by a mass as large as a duck's egg and of a similar shape. The mass was tense, fluctuating, and smooth, and that portion of the gut wall opposite the mesocolic attachment was freely movable over it. The mass was at first supposed to be an intussusception of the terminal por-

tion of the ileum, but as no reduction could be effected by taxis or traction on the ileum, the cæcum was opened by an incision parallel to its circular fibres, and the tumor delivered and examined. It was covered by what appeared to be congested, eroded and transformed mucosa. It was apparently sessile over about one-fifth of its surface, that portion being attached to the mesocolic border. (Here, and subsequently, I use the terms "mesocolic" and "mesocolon" for the sake of convenience, to indicate that portion of the ascending colon which lies in contact with the parieties, and is devoid of a peritoneal covering. This is the manner of fixation which is regarded as normal in 74 per cent. of cases.*)

The examining finger was then passed around the lower extremity, which lay free in the cavity of the cæcum, and on through the ileocæcal valve, which was found to be patulous. The upper half of the ileocæcal valve was apparently fused with the outer covering of the tumor. The upper end, corresponding to the smaller end of an egg, was more sessile than the lower, and terminated in a sort of funicular process, about two inches in length, lying in contact with the mesocolic wall of the gut, its diameter diminishing to nothing at its upper end. An incision was then made through the presenting wall of the tumor, in the line of its longest diameter, and between three and four ounces of a clear, viscid fluid was evacuated, which, unfortunately, was not saved.

The cavity of the cyst was then inspected and was found to be lined with a smooth, glistening membrane, of a grayish color The cyst wall was three-sixteenths of an inch in thickness, firm and hard, but somewhat friable on its outer surface. The interior lining was smooth and very firm, and the cavity of the cyst corresponded in shape to its outer surface, with the exception that it did not extend into the funicular process, already described, at the upper end. While at the site of the attachment of the tumor to the gut wall there was discovered a funnel-shaped pocket extending for about two inches backward and inward between the layers of the mesentery of the ileum, and just large enough to easily admit the examining finger.

No attempt was made to enucleate the cyst on account of the firmness of its incorporation with the adjacent structures. The only procedure by which the cyst could have been removed would have been a complete resection of the cæcum, the terminal por-

* Huntington, Anatomy of Peritoneum and Abdomen, p. 82.

tion of the ileum, and part of the ascending colon, and this was not considered advisable. Consequently, the free portion of the cyst wall was trimmed off to within a quarter of an inch of its point of fusion with the gut, and the somewhat sharp venous hemorrhage from the cut surfaces was readily controlled by a rapidly applied continuous catgut suture. The wound in the gut was then closed by three layers of sutures, applied in the usual manner. A small cigarette drain was left in as an extra precaution, and the abdominal wound was closed in the usual way. The drain was removed on the third day, and the patient made an uninterrupted recovery.

To my great chagrin the excised portion of the cyst wall was lost, and in the absence of any pathological report I am reduced to the necessity of speculation to determine the character of this most rare condition. A few cases have been described which resemble this one to a limited extent, but upon close study their points of similarity with this one are so few as to render them of little value in determining the origin of the cyst, and therefore my conclusions are based rather on analogy than on the reports of the cases themselves.

In order to arrive at a definite conclusion as to the origin of the cyst reported above I shall review briefly the question of cysts of the intestine, mesentery and general abdominal cavity in the light of recent pathological investigations. The subject resolves itself, practically, into two classes: Cysts of the gut itself, and cysts of the mesentery. The question of cysts of the gut is not a large one; that of mesenteric cysts is more complex and will require a few words of preliminary explanation.

In 1892 Bracquehaye[7] classified mesenteric cysts as follows:

1. Sanguineous cysts, or hæmatomata, including certain serous cysts.

2. Lymphatic cysts, including chylous cysts and most of the serous cysts.

3. Parasitic cysts; hydatids.

4. Congenital cysts, or dermoids.

5. Cysts of adjoining organs (ovaries, parovarian, head of pancreas, etc.).

In 1897 Moynihan[49] classified them as:

1. Serous cysts.

2. Chyle cysts.

3. Hydatid cysts.

4. Blood cysts.

5. Dermoid cysts.

6. Cystic malignant disease.

In 1900 Dowd,[19] finding these two classifications unsatisfactory, made a further classification based upon the origin of the cysts, which, while simpler than those quoted above, seemed also to be more reasonable.

Dowd's classification is as follows:

1. Embryonic cysts.

2. Hydatid cysts.

3. Cystic malignant disease.

It will be seen that Dowd has eliminated such terms as "sanguineous," "lymphatic," "serous" and "chylous" in his classification of these cysts, for reasons which he has set forth in his admirable paper. As his arrangement seems to me to be far more rational and scientific than the earlier ones, it may not be amiss to give here briefly his reasons for the eliminations he had made. The Bracquehaye and Moynihan classifications are essentially similar, and a criticism of the headings of the former which he has eliminated will indicate sufficiently the weak points of both.

1. *Sanguineous Cysts*—This term is misleading, since blood-containing cysts of the mesentery may be either simple hæmatomata or any variety of cyst into which hemorrhage has taken place secondarily. Cysts have been described under this heading in which the contents varied from "slightly blood-stained" (Morton[49]) to "thin brownish-red" (Hahn[28]), though the evidence is in favor of the belief that they were not primarily blood cysts at all, but were cysts of some other nature into which hemorrhage had subsequently taken place. I agree with Dowd that these cysts should be classified according to the structure of their walls and the chemical character of the fluid, rather than to put them under the head of blood cysts merely on account of the apparent character of their contents.

2. *Lymphatic Cysts (Chylous or Serous)*—Although for many years authors have employed these designations in their description of certain mesenteric cysts, the propriety of such a terminology seems to me to be open to considerable doubt, and,

as expressing my own conviction on this point, I cannot do better than to quote from Dowd's article on mesenteric cysts.*

"It has been generally supposed that these [chylous] cysts are due to a dilatation of some one of the lacteal or the chyliferous vessels, as taught by Rokitansky half a century ago. It has also been suggested that there has been an effusion of chyle into previously existing cysts; this seems much more probable. We can see how a duct which has a gland behind it might become cystic if it were occluded; the size of the cyst depending upon the extent to which the pressure of the cyst fluid could distend its wall. It is not easy to understand how this process could take place in vessels so rich in anastomosis as are the chyle vessels. There are records of at least ten cases where the thoracic duct, or one of its main branches, has been wounded, and either sutured ligated or packed, without evidence of cyst formation. Ziegler[74] states that occlusion of the thoracic duct may be followed by anastomosis, or by distention of the lymph vessels, and mentions the distention of the lacteals into elongated, tortuous forms in certain instances where they have been obstructed.

"One cannot deny the possibility of cyst formation by this process, but it is unlikely, and at variance with ordinary pathological processes. It seems far more likely that chyle should be effused into the cavity of cysts already formed, particularly as lymph nodes have been found in the walls of mesenteric cysts, thus indicating the presence of lymphatic structures; and spaces are seen in the cyst walls which are apparently lymph channels.

"Taking these cases together, it seems pretty well established that chylous cysts are really preformed cysts, situated in such close relation to the lacteals that chyle has been effused into them, and that they are really of embryonic origin, in structure similar to ovarian and parovarian cysts."

The etiology of cysts within the abdominal cavity is at best a complex subject, and in order to make the question more readily comprehensible, I have adopted the following classification, which is based on Dowd's arrangement, but enlarged to include cysts of the intestine itself. There are also some minor amplifications which have been suggested by reports of recent cases:

1. Cysts arising from the glandular structures of the intestinal wall.

2. Parasitic cysts (hydatids).

* Annals of Surgery, October, 1900.

3. Embryonic cysts.

4. Cysts of the normally placed retroperitoneal organs.

5. Cystic malignant disease.

I will consider them as briefly as possible in the above order:

1. *Cysts Arising from the Glandular Structures of the Intestinal Wall*—These cysts originate in an occlusion of the mouths of the intestinal glands (crypts of Lieberkühn, etc.), following some inflammatory process, generally a chronic enteritis or colitis.

The inflammation and subsequent thickening of the mucous membrane closes the mouths of these follicles, the cavities of which then become cysts by the retention of their secretions (Fränkel[23]). These cysts are generally multiple and vary in size from 1 to 4 cm. in diameter. They may occur in any part of the colon or small intestine, but are generally found in the former.

Rokitansky[58] says: "Cysts of the intestinal wall are rarely seen. The cases mentioned in this relation are principally multilocular, partly lying in the gut wall and partly in the subserous tissue, and containing serous fluid."

Fränkel[23] reports the case of a four-year-old child upon whom a resection of the head of the femur had been done for osteitis. Convalescence was interrupted by an attack of dysentery, followed by death. The post-mortem showed a thickening of the mucous membrane of the intestine, and many small cysts the size of cherries lying between the mucosa and serosa. Chemically the fluid contents consisted of mucin. The cysts lay principally in the mucous membrane, projecting downward to the muscular wall and upward to the muscularis mucosæ. The neighboring follicles of Lieberkühn showed the process of cyst formation from compression of their outlets. This was caused by a small-celled infiltration of the epithelium accompanied by a loss of the normal epithelium.

Another case reported by the same observer probably also belongs under this heading. The patient was an infant that died on the third day after birth with symptoms of intestinal obstruction. At the autopsy a cystic tumor was found at the end of the ileum. It was round, 2½ cm. in diameter, and projected into the lumen of the ileum and cæcum. It was not perceptible from the outside. The ileocæcal valve was stretched over the cyst, which contained clear mucus. A microscopic examination was not made, but macroscopically the inner wall appeared to be formed from the muscularis mucosæ.

Krogius,[39] in a review of the post-mortem findings in sixteen cases of enteric cysts, says in part: "In four instances the cysts had their origin on this valve (the ileocecal). Most of the cysts were in the intestinal wall itself, presenting on the convex side of the gut, and apparently sprung from either the submucous or subserous structures, or from between the two muscular layers. *Only in a few instances have they been found on the concave surface, between the layers of the mesentery.* A communication between the cavity of the cyst and the lumen of the gut existed in only two cases; in all the remaining cases the cyst cavities were entirely shut off from the canal."

An understanding of the morphology of the submucosa will indicate the possibility of retention cyst formation in that structure, although it is doubtful if cysts originating here could be differentialted macroscopically from cysts of the mucosa unless they were of very small size.

Huntington[35] says: "Situated between the mucous and muscular coats of the intestine this layer is known as the *submucosa* It contains, embedded in its tissue, the glandular elements of the intestine, derived from the entodermal epithelium, and the blood-vessels, lymphatics, and nerves."

2. *Parasitic Cysts (Hydatids)*—These may occur in the mesentery, as well as in other parts of the body, and should be considered as primary in the structure in which they are found. I have no record of one having been reported in the intestine itself, but Nannotti[50] has collected twenty-nine cases of hydatids of the mesentery. Psaltoff[55] and others have made similar observations.

This form of cyst is due to the growth of the *tænia echinococcus*, and the diagnosis is generally made by the peculiar, laminated character of the cyst wall and by the finding of scolices or hooklets, or both, in the cyst cavity. Sterile cysts are often found in which the scolices are absent. The normal echinococcus fluid is clear or slightly opalescent, though sometimes rendered turbid from admixture with disintegrated scolices, or fragments of the parenchymatous layer. It is either neutral in reaction, or slightly acid, or slightly alkaline, with a specific gravity between 1.000 and 1.015. It contains no albumin, but does contain sodium chloride, succinic acid, ptomains and toxins.

3. *Embryonic Cysts*—(*a*) From remains of fetal organs; Wolffian and Müllerian bodies, omphalomesenteric duct. (*b*) From snared-off portions of the intestine; also Remak's diverti-

culum, and atypical intestinal diverticula. (*c*) From sequestrations of portions of the various retroperitoneal organs. (*d*) Dermoids.

Under this heading belong the major part of the cysts found in the abdominal cavity. Warthin[70] says: "Cysts arising from misplaced or persistent entodermal or mesodermal anlage are of relatively frequent occurrence. They are found most frequently in the broad ligaments and tubes, less often in the peritoneal cavity, intestine, in the neighborhood of the trachea and bronchi, in the lungs, pleura, tongue, neck, liver, kidneys, etc.

"They owe their origin to the persistence of fetal glands or ducts, or to misplaced entodermal or mesodermal epithelial anlage. Those found in the uterine wall, broad ligaments, and tubes arise from remains of the Wolffian body or the duct of Gärtner. In the peritoneal cavity and abdominal wall they may arise from snared-off and persistent portions of the intestine (enterocysts), or from portions of the urachus (urachus cysts). In the liver and kidney they may arise from portions of the gland tubules, which become constricted during the period of development (adenocysts).

"In the abdominal wall cysts lined with columnar cells may arise from remains of the omphalomesenteric duct (omphalomesenteric cysts)."

In this connection it may not be inappropriate to mention those extremely rare congenital cyst tumors of the abdomen which contain a more or less well-developed fetus, alive or dead.

Himly,[32] in an autopsy upon a female child, aged two and a half years, found a cystic tumor in the epigastrium, which contained a fairly well-developed fetus, with, however, defective extremities and head.

Klebs[37] reported the case of an infant in whom independent motions were observed within an abdominal tumor. The child died a few weeks after birth, and at the autopsy a cyst was found behind the transverse colon, composed of peritoneum and amnion, and containing an umbilical cord to which was attached a partially developed fetus.

Highmore[31] reported the case of a boy, aged seven years, in whose abdomen a cystic tumor began to develop. Its increasing size caused repeated attacks of partial intestinal obstruction, and at the age of fifteen years he died in consequence of an intestinal hemorrhage. During life pulsations could be felt in the cyst, and

the patient declared that his abdomen contained something living. The autopsy disclosed a tumor weighing over four pounds, attached along the entire length of the duodenum, which communicated with it and formed part of the sac. The latter contained an imperfect fetus, the nutrition of which was maintained by a short cord which established the connection with the vascular walls of the sac. (Piersol, *Reference Hand-book of the Medical Sciences*, "Teratology.")

(*a*) *From Remains of Fetal Organs*—The part played by these remains in the production of abdominal cysts is very clearly set forth in the following case reported by Lockwood:[42] The patient was a female, aged twenty years, in whom an operation disclosed a cyst as large as an ostrich's egg, behind the descending mesocolon. The colon lay to its outer side, but had no connection with it. After removal of the cyst the ureter, which lay behind it, came into view. The cyst itself lay among loose connective tissue behind the peritoneum. It was unilocular, and filled with a chocolate-colored, semi-fluid mass, consisting of fibrin, blood pigment and cholesterin. Histological examination showed the contents to be simply an altered blood clot. It did not show either scolices, hooklets, or daughter cysts. There was no glandular structure found. In attempting to account for the origin of the cyst, he says, in part:

"The suprarenal body may be developed from the Wolffian body, but at all events it is continuous with it until a late period of intrauterine life. At this period the lower end of the Wolffian body is already becoming converted into the epididymis and other structures about the testis; in the female it is becoming epi- or parovarian. Thus it is evident that between the suprarenal body and the ovary or testis there is, along the course of the ureter, a part of the Wolffian body which has hitherto been unaccounted for. How long this persists or what may be its ultimate fate is unknown. * * * Now, it is so notorious that the part of the Wolffian body which is in relation to the ovary or testis is prone to give rise to various kinds of cysts, that it does not seem unreasonable to assume that the remaining part—viz., that which lies along the course of the ureter—may do so likewise; and I venture to suggest that this is the origin of the cyst which I have just described."

According to Moynihan,[49] the congenital remnants of the Müllerian and Wolffian ducts and bodies are regularly found be-

tween the layers of the mesentery; and it is now very generally believed by pathologists that these "rests," which seem particularly prone to undergo cystic degeneration, are responsible in a great measure for certain mesenteric cysts, as well as for cysts of the ovary, parovarian, and Gärtner's duct.

The origin of the congenital cystic kidney is by some pathologists ascribed to an inclusion of a portion of the Wolffian body or Müller's duct within the kidney itself. (Von Bergmann,[68] Sutton.[63])

Dowd, referring to this method of causation, says: "It is altogether within the bounds of probability that such a separation should from time to time take place from the Wolffian body or the germinal epithelium at an early time in embryonic life; and, if such portions are separated, it is not strange that they should be carried into the mesentery, mesocolon, or mesorectum in the course of their development, and there form cysts * * *; or, if they should not be carried into the mesentery, they might develop as retroperitoneal cysts, some of which are certainly similar in structure to the mesenteric cysts; or, if they should be carried into that portion which forms the great omentum, they would form omental cysts, which are also of a similar structure."

The omphalomesenteric duct is another fetal organ which may give rise to intra-abdominal cysts. Riesman,[57] speaking of Meckel's diverticulum, says: 'It is usually on the convex border of the gut, but it sometimes arises nearer the mesentery. Very rarely it lies entirely within the mesentery." Its usual position, with regard to the ileocæcal valve, is about one metre or more above that structure, but Fitz[22] reports a case of his own in which the obliterated remains of the omphalomesenteric duct were found at a distance of 3 cm. from the valve; and Wernher[72] cites another instance in which it was found at a distance of an inch and a half from the cæcum.

Piersol[53] says in this connection: "The diverticulum may become constricted and its communication with the gut entirely lost, it remaining attached to the gut as a cyst-like appendage. The latter may mark the seat of an active growth, resulting in the production of an intestinal cyst of huge size."

The following curious case, reported by Poirier,[54] was probably of this origin: The patient was a woman aged twenty-eight years, who had had repeated attacks simulating appendicitis. An operation disclosed a cystic tumor the size of an orange, attached

to the convex side of the terminal portion of the ileum by an imperforate pedicle, the torsion of which was responsible for the attacks.

Krogius[39] quotes Quesnal as follows: "In sixteen cases of simple intestinal cysts ten were in children less than six months old, four in children from one to five years old, and in only two cases were the patients older (one twenty years old and one sixty years). They occur with about the same frequency in males and females. As a rule, the cysts have been small, from the size of a cherry to 4 cm. in diameter. *The cysts were generally found at the site of the usual location of Meckel's diverticulum* in the lower part of the ileum, the greatest distance above the *valvula Bauhini* (ileocæcal valve), being one metre."

I have been unable to find records of any cases which were ascribed positively to an occlusion of the lumen of Remak's diverticulum, but I think it possible that some of the cases reported above may have been of this origin.

This diverticulum originates from the epiblastic layer of the neural canal in very early fetal life, owing to a faulty closure in this canal a process of the epiblastic layer may be pushed forward through the chorda dorsalis and between the layers of the primitive mesentery. With the subsequent closure of the canal the extruded portion is snared off and remains as a potential cavity lined with epithelium, which may eventuate in a retroperitoneal or mesenteric cyst.

(*b*) *From Snared-off Portions of the Intestines*—The possibility of this etiology has already been briefly alluded to above. Dowd[19] has collected the reports of four of these remarkable cases, which I quote below:

"The most remarkable one was recorded by Studgaard.[64] It was in the mesentery, contained 200 c.c. of chocolate-colored fluid, and had a funnel-like process or pedicle thick as one's thumb, which had to be severed in removing the cyst, the cut extending through the different layers of the cyst wall. The inner surface of the sac was in parts smooth and shiny; another part had the appearance of mucous membrane, and section through this showed a layer of tubular glands tightly packed together, with a membrana propria, a single layer of cylindrical epithelial cells, and a perceptible lumen; beneath this layer there was a muscularis mucosa, then a submucous layer of connective tissue, then a layer 2 mm. in thickness, composed of smooth muscle fibres

running parallel with the circumference of the aperture in the cyst wall; and outside of this another layer, half as thick, composed of smooth muscle fibres running at right angles with those of the first layer; then connective tissue and serosa.

"The cyst wall was therefore in perfect agreement as to structure with the wall of the intestine, save that in the latter we do not find an irregular hypertrophy of the tubular glands, with degeneration of the superficial layer which was shown here, nor were Peyer's patches or solitary glands shown. On the smooth portion of the cyst wall this layer of villous mucous membrane was represented by a connective-tissue layer containing pigment and epithelial cells, irregular in formation and arrangement. The situation in the mesentery is not given.

"Eve[20] has described a cyst removed from the mesentery of the jejunum, which had in its wall three layers of unstriped muscle fibres. The section showed the middle layer cut transversely and the other two longitudinally. He refers to specimens in the Museum of the Royal College of Surgeons, No. 2352 E, from a girl of nine, which had a considerable quantity of unstriped muscle fibre in its walls.

"Fehleisen[21] has also described a multilocular cyst which contained 8,200 c.c. of clear, reddish-brown fluid, and had unstriped muscle fibres in the cyst walls. The walls contained three layers: 1. An outer, dense connective tissue. 2. Median, very vascular connective tissue, loosely arranged. 3. Internal layer, exclusively unstriped muscle fibres, arranged longitudinally, but with large bundles having a more or less irregular construction. 'The muscle fibres were characteristic; they differed in no respect from those found in the intestinal tract or bladder.'

"Brentano[8] also describes a mesenteric cyst in the wall of which there were smooth muscle fibres under the serosa. The fluid contained fibrin and degenerated blood cells."

Although I have placed cysts under the heading of "Snared-off Portions of the Intestine," to conform to the classification given them by the pathologists, I am inclined to attribute them rather to snared-off portions of some of the various atypical diverticulæ, of which I will now speak.

Multiple diverticulæ of the large and small intestine have been frequently reported, and although I can find no record of their having given rise to cysts, it is not difficult to believe that they might do so under favorable conditions.

Wallman[69] says: "Diverticulæ of the intestine are not uncommon. In the Josephs Academy Museum of Pathological Anatomy is a piece of small intestine, 48 cm. long, containing thirty-seven diverticulæ ranging in size from a bean to a pigeon's egg. *Thirty of these lay between the layers of the mesentery.*"

He also described a true diverticulum, 10 cm. long, located on the free surface of the large intestine, two feet above the ileo-cæcal valve. Another specimen in the same collection, a piece of small intestine, has on its free surface two diverticulæ, one 8 cm. and the other 11 cm. long.

In an autopsy on the body of a man, aged sixty-six years, he found nine true diverticulæ of the large intestine distributed as follows: One on the ascending colon, three on the transverse, three on the descending, and two on the sigmoid flexure. Of these, seven were on the free surface, the other two being between the layers of the mesocolon. Grawitz,[26] in an elaborate article, refers to this condition and reports several cases.

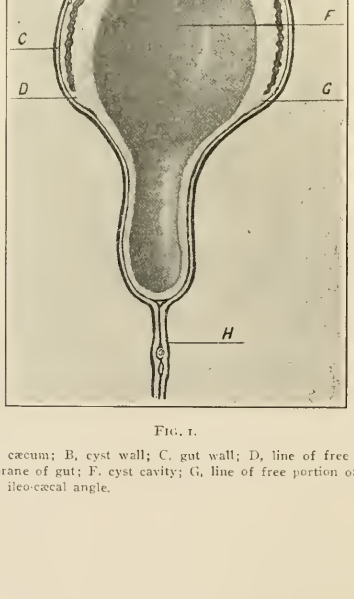


FIG. 1.

A, cavity of cæcum; B, cyst wall; C, gut wall; D, line of free portion of cyst; E, mucous membrane of gut; F, cyst cavity; G, line of free portion of cyst; H, leaves of peritoneum at ileo-cæcal angle.

(*c*) *Cysts Arising from Sequestrations of Retroperitoneal Organs*—The literature of teratology is replete with instances of this curious sequestration of portions of the various organs in all parts of the body, and it is well recognized that such fragments are prone to undergo cystic degeneration in their abnormal locations.

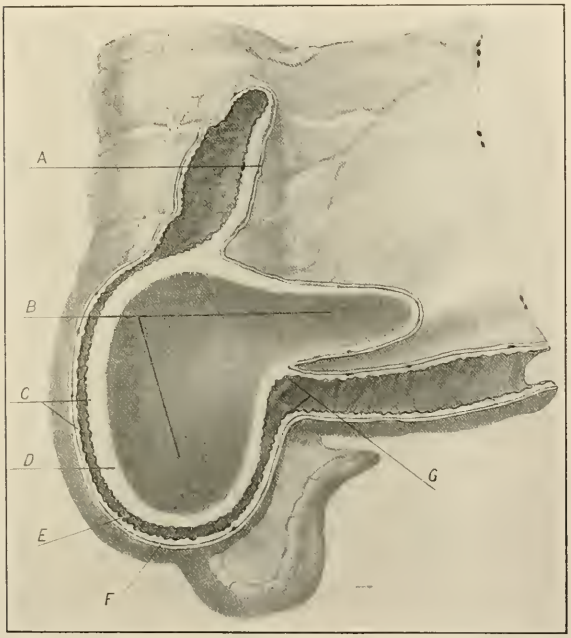


FIG. 2.

A, funicular process; B, cyst cavity; C, mucous membrane; D, cyst wall; E, cavity of cæcum; F, cut edge of gut wall; G, ileo-cæcal valve.

Piersol[53] makes the statement that accessory or dislocated lobes of the liver have been found within the falciform ligament; that isolated masses of pancreatic tissue have been found embedded within the intestinal and gastric walls. These masses may remain in *statu quo*, or may later in life become the seat of cysts

of large size. Fitz[22] refers to two cases where an accessory pancreas was found in the end of Meckel's diverticulum.

Accessory spleens have frequently been described. Albrecht[1] describes a case in which the principal organ was absent, and instead segments or supernumerary bodies of splenic tissue to the number of almost 400 were found scattered throughout the abdomen.

Accessory suprarenals have been found at considerable distances from the main organ; for instance, between the testicle

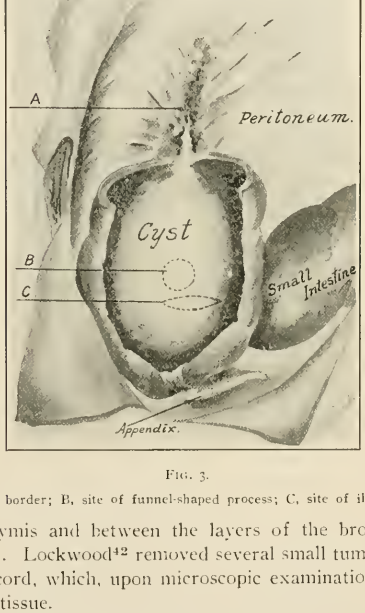


FIG. 3.

A, mesocolic border; B, site of funnel-shaped process; C, site of ileo-cæcal valve.

and epididymis and between the layers of the broad ligament (Piersol[53]). Lockwood[42] removed several small tumors from the spermatic cord, which, upon microscopic examination, proved to be adrenal tissue.

Cases of congenital fragmentation of the *glandular* elements of the prostate have also been observed.

(*d*) *Dermoids*—I can find no record of retroperitoneal dermoids in the male, though numbers of cases have been reported

in the female. It is very generally believed that these tumors have their origin either in a sequestration of ovarian tissue or in a developmental defect of an ovum. Douglas,[18] however, believes that they may originate in structures which persist from fetal life, as the Wolffian body and Müllerian duct. Dermoids of the testicle have been reported, and as the testicle is originally a retroperitoneal organ, it is conceivable that cryptorchism, complicated by dermoids, might occur. These dermoids are thought by some pathologists to be due to a double fecundation.

Cohenheim attributed the origin of retroperitoneal dermoids to the inclusion of aberrant portions of the ectoderm within the ovary in early fetal life, and for a long time it was believed that all mesenteric dermoids had an ovarian origin, and only secondarily became mesenteric (Bauman[4]).

Hall[29] has recently reported a case of a large dermoid cyst of the mesentery of the ileum in a girl aged eight years. Its position was twenty-five inches above the ileocæcal valve. Other cases have been reported by Schutzer,[62] Mayer,[45] Spencer Wells,[71] Langton,[40] and König.[38]

4. *Cysts of the Normally Placed Retroperitoneal Organs*—Instances of these cysts are too numerous to require more than a word in passing. The organ most likely to give rise to a mesenteric cyst is the pancreas. Hodenpyl[34] tells me that he recently discovered such a cyst post-mortem which had pushed its way for some little distance between the layers of the transverse mesocolon.

5. *Cystic malignant disease* of the mesentery or intestine is of rare occurrence. Dowd cites a case reported by Keen[36] of a cystic sarcoma of the omentum in which the fluid weighed 10 pounds. Graff[25] removed part of a dermoid cyst as large as a child's head, which presented in the ischiorectal fossa, but which had its origin between rectum and sacrum and had begun to undergo carcinomatous changes.

Diagnosis of the Nature and Method of Production of the Cyst in Question—In the absence of any histological details I am obliged to arrive at my conclusions by a process of elimination, based largely upon the gross characteristics of the cyst.

In the first place, from the presence of the curious diverticulum of the cyst which lay between the layers of the mesentery of the ileum (or behind the reflexion of parietal peritoneum on the inner side of the colon), I conclude that the cyst was either of

mesenteric or retroperitoneal origin. This and the thickness and density of its walls would at once exclude cysts originating from the mucosa or submucosa of the gut. A hydatid origin may next be excluded with a fair degree of certainty by the viscid clearness and homogeneity of its fluid contents. The same reasons would also exclude a dermoid. Cystic malignant disease is excluded, for

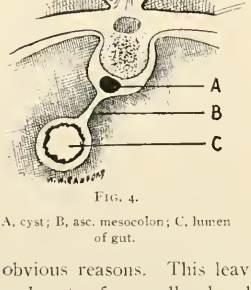


FIG. 4.

A, cyst; B, asc. mesocolon; C, lumen of gut.

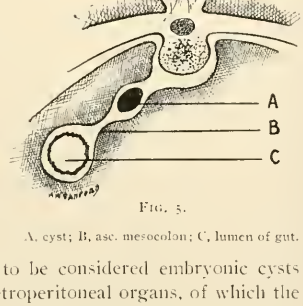


FIG. 5.

A, cyst; B, asc. mesocolon; C, lumen of gut.

obvious reasons. This leaves to be considered embryonic cysts and cysts of normally placed retroperitoneal organs, of which the latter can be safely thrown out, as there is no organ in the immediate vicinity which could give rise to such a cyst.

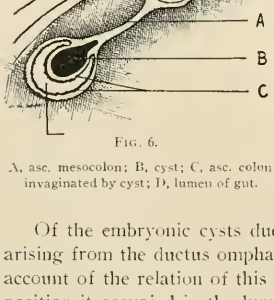
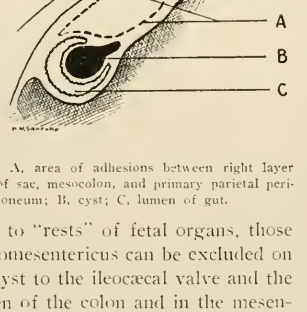


FIG. 6.

A, asc. mesocolon; B, cyst; C, asc. colon invaginated by cyst; D, lumen of gut.



A, area of adhesions between right layer of sac, mesocolon, and primary parietal peritoneum; B, cyst; C, lumen of gut.

Of the embryonic cysts due to "rests" of fetal organs, those arising from the ductus omphalomesentericus can be excluded on account of the relation of this cyst to the ileocæcal valve and the position it occupied in the lumen of the colon and in the mesentery.

An origin from a snared-off portion of the intestine can, I think, be excluded, as the walls of this cyst had nothing suggestive of this condition. The same reasoning also applies to cysts arising from Remak's diverticulum or from the atypical diverticulæ heretofore described.

Between the two remaining varieties of embryonic cysts, those due to fetal "rests" and those due to sequestrations, I am unable to decide with so much certainty, but I am inclined to throw out the latter on the ground of probability, in the absence of a better reason, because I believe that the "rests" above referred to are a more frequent source of cysts than are the sequestrations.

For the sake of argument, then, let us suppose that the cyst in question had its origin in the Wolffian body, the Wolffian duct, or Müller's duct. A more accurate designation as to which of these structures was concerned in its production is impossible, as well as immaterial. Recognizing the tendency of these "rests" to undergo cystic degeneration and postulating the existence of such a process in the present instance, it is not difficult to conceive the chain of events beginning with a tiny retroperitoneal "rest" *in fetu* and ending with the same structure, which has undergone cystic degeneration, has become many times enlarged and has migrated between the layers of the mesentery during the development of that structure as far as its attachment to the gut, there pushing before it the structures forming the wall of the intestine, and eventually lying, to all intents and purposes, within its lumen, though anatomically speaking, outside it. Such a cyst, a portion of it lying between the comparatively unyielding layers of mesentery, and another portion lying free within the lumen of the intestine, would be likely to assume the pyriform outline which I have described as being one of the peculiarities of the cyst in question. The position of the cyst at the angle formed by the mesentery of the ileum and the reflexion of the peritoneum at the ascending colon renders it difficult to say under which of these structures the funnel-shaped portion lay; but this is a matter of no consequence, since the mesentery and this peritoneal reflexion are, in early fetal life, one and the same anatomical structure, and it is only after the rotation of the intestines and the subsequent adhesion between the right leaf of the mesentery with the parietal peritoneum that the differentiation between mesentery and ascending mesocolon becomes an anatomical fact.

(I use here the term "mesocolon" merely as a matter of convenience, and in the same sense with which I have qualified it earlier in this paper, in order to indicate that portion of the ascending colon which is normally adherent to the parieties in the adult, and to convey to the mind the relation which this portion bears to the ascending mesocolon in the fetus.)

Figs. 4, 5, 6, 7 and 8 are intended to represent in a schematic way the probable course and final resting-place of the cyst which I have described, and will, I think, require no further elucidation.

As I have stated at the beginning of this paper, I have been unable, after a fairly careful search through the literature of en-

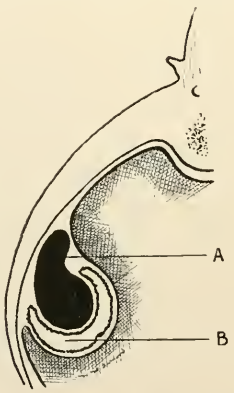


FIG. 8.

A, cyst; B, lumen of cæcum invaginated by cyst.

teric and mesenteric cysts, dating back many years, to find any record of a cyst similar to the one here reported. I have, however, found reports of several enteric cysts of comparatively large size, though evidently not of the same origin as this one, and, as a matter of interest in this connection, I give below a condensed description of the more appropriate cases.

In 1887 Sainsbury[60] removed from the cæcum of a young girl a cystic tumor the size of a duck's egg, ovoid and smooth. Anatomical and histological details are wanting. The contents were

a very dark, ropy, mucoid fluid. He believed that it was possibly due to a cystic dilatation of one of the lips of the ileocæcal valve.

In 1903 Condamin[14] operated upon a woman for intestinal obstruction. Upon opening the cæcum, within which a mass was felt, he found a cyst filling up the entire cavity of the gut, and attached by a pedicle to the wall near the origin of the appendix. He states that the patient had previously been operated upon for a right ovarian cyst. The histological appearance of the specimen removed resembled the structure of a typical ovarian cyst.

In 1856 Conant[13] reported having found at autopsy in a male subject a cystic tumor containing a thick, creamy, semi-fluid substance and "attached to the ileum at its junction with the cæcum. It appeared to have existed between two folds of peritoneum." This is the only case I have been able to find which in any way resembled my own, but the details are so vague as to render it of little value.

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MULTIPLE SYSTEMIC SARCOMA OF MYELOGENOUS ORIGIN.*

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The case I am about to present was that of a patient in the wards of Bellevue Hospital, in the service of Dr. Egbert Le Fevre. I am greatly indebted to Dr. Le Fevre for permission to present at this time an abstract of the clinical history. The case will be reported in full later, with Dr. Levre.

The patient was a boy, sixteen years of age, a piano player, of good habits, with no history of previous illness or of venereal disease. The family history was likewise good.

The patient had been losing weight for an indefinite time. Six weeks previous to admission, May 3, 1906, he noticed a few nodular swellings behind the angle of the jaw, and also that he was gradually becoming weaker. Later, he became aware of a mass in the left side of the abdomen as well as of nodules about the size of a pea in the abdominal wall, which were movable and not tender. No other symptoms, except daily increasing weakness, were elicited. His appetite was good. A week before admission he complained of pain in the left hip, which radiated down the thigh. Since then the pain had been continuous in the left knee and hip, without any swelling of the joints.

On admission his temperature was 101.4°; pulse, 120; respiration 20. Patient was a boy of small frame, and poorly nourished. The skin was pale; the mucous membranes anæmic. There was a small discharging pustule over the left buttock. The lymph nodes in the neck were greatly enlarged. All the superficial lymph nodes were easily palpable and firm. In the subcutaneous tissues of the neck there were found a few large masses which were irregularly nodular, movable, and not attached to the skin. Numerous small nodules were felt through the skin of both upper arms, and in the epigastrium. Above Poupart's ligament on the left side was felt a firm mass about the size of a hen's egg, which

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was not attached to the abdominal wall. The rounded border of the liver was felt indistinctly about one inch, the spleen about three inches, below the costal arch.

On May 7, four days after admission, one of the subcutaneous nodules was removed and examined outside the laboratory, and I am informed that the diagnosis of alveolar sarcoma was made.

Two weeks after admission the patient complained of pain and tenderness and pressure in the left axilla, and a few friction rales were heard.

On May 18, the next day, another small subcutaneous nodule was removed and examined by us.

The patient died suddenly on May 19, sixteen days after admission.

The urine was examined twice, and was found negative. There is no record of the presence of the Bence Jones albumin in the urine, the test probably not having been made.

Two examinations of the blood were made. The first day after admission the red blood cells were 3,940,000; white cells, 8,600; hemoglobin, 55 per cent. The red blood cells showed poikilocytosis and central pallor. A few normoblasts were present. Two weeks later, three days before death, the red blood cells were 2,800,000; white cells, 8,600; hemoglobin, 45 per cent.

One blood culture, made eight days before death, was negative. The patient ran a daily evening temperature of 100°. The last two nights it ran to 101°.

After considerable difficulty, consent for an autopsy was obtained and this was performed fifty hours after death. Only a brief abstract of the autopsy findings need now be given.

The various organs showed a slight degree of post-mortem change. The body was that of a boy of medium stature, markedly emaciated. Numerous shot-like nodules were felt in the subcutaneous tissue of the neck and thorax, and there were a few on the back, abdomen and upper extremities, none being present in the lower extremities. The lymph nodes in the axillary and inguinal regions were not enlarged.

The peritoneal cavity contained about three liters of serofibrinous fluid. The omentum presented a very striking picture, appearing as a large apron covering the intestines. In places it was at least two cm. in thickness. The spleen extended below the umbilicus; the liver several inches below the free margin of the ribs. On the removal of the sternum and cartilages, which were normal in appearance, a series of small, firm nodules, two

to three mm. in diameter, were found, adherent to their inner surfaces. All the cervical lymph nodes, the superficial as well as the deep chains of nodes, were enormously enlarged, forming large, more or less adherent packets on either side of the trachea and thyroid. In general, the individual nodes were discrete, but were firmly matted together with adhesions. In places the outlines of the individual lymph nodes were not sharply delineated, the nodes being more or less intimately fused together. The largest nodes were situated along the carotids, just above the bifurcation, and measured roughly 5 x 3 x 2 cm. The smallest nodes were whitish gray and, on section, homogeneous, permitting no juice to be scraped off. The larger nodes showed central yellowish areas of necrosis, some of which were softened. In other places there were irregularly shaped areas of hemorrhage and hemorrhagic softening.

The thyroid gland preserved its normal contour, measuring 6 cm. in breadth. Many whitish nodules and in places, larger masses were present throughout the gland. Only a small portion of normal tissue was apparent. In the region of the thymus there was a large mass of tissue measuring 10 x 8 x 4 cm., extending over the pericardium. It was composed of a series of larger and smaller nodules. No thymus-like tissue was discernible on section.

The pleural cavities contained about a liter of sero-fibrinous fluid. The costal and diaphragmatic pleuræ were studded with numerous elevated, whitish, firm nodules varying in size from a pinhead to the size of a large pencil, and hard, flattened, but elevated plaques. The visceral pleuræ were likewise studded with nodules, some of which were flat and sessile, others more or less pedunculated. No nodules were present in the lung tissue proper, or in the bronchial nodes, both of which were free from tuberculosis. The tracheal nodes, however, presented large tumor masses.

Heart—The mediastinal surface of the pericardium presented a series of nodules resembling strings of smaller and larger pearls. A few raised nodular foci were found upon the coronary veins. The interauricular septum was thickened with tumor tissue, and minute nodules were present in the papillary muscles of the left ventricle.

Liver—The liver weighed 2,090 grams. It was thickly studded with tumor masses varying considerably in size, shape, and gen-

eral appearance. In general, they were sharply circumscribed, some of the nodules being surrounded by a translucent fibrous capsule.

Spleen—The spleen was very large, weighing 1,080 grams, and measuring 21 x 16 x 9 cm. Like the liver, it was the seat of extensive tumor infiltration.

Adrenals—The adrenals weighed 55 grams together. They were studded with tumor nodules, little normal tissue being present.

Kidneys—The kidneys together weighed 350 grams. The capsules on their external surfaces were found studded with large, confluent nodules, also flat foci, 1 to 2 mm. in size, and raised nodules of intense purple color, 3 to 4 mm. in diameter.

The left testicle was situated within the peritoneal cavity in a mass of enlarged lymph nodes. The organ was infiltrated with hemorrhagic and softened tumor tissue.

The periportal lymph nodes were greatly enlarged. The peripancreatic lymph nodes were infiltrated. The pancreas presented a few small tumor nodules.

The entire alimentary canal, from the mouth to the anus, especially with regard to the mucous membrane, was normal. Along the mesenteric attachment of the small and large intestines were found innumerable smaller and larger spherical nodules, varying greatly in size, the largest being about the size of a large lime. Similar nodules and plaques were scattered along the serosa of the mesentery. The mesenteric and retroperitoneal lymph nodes were all greatly enlarged. As in the case of the cervical lymph nodes, these nodes, although adherent, retained their normal shape, and in only a few places had the individual nodes become confluent.

Head—The pericranial tissues, especially on the right side, were thickened, large flat masses of tumor-like infiltration being present over the convexity. The parietal bone over the boss was eaten away or depressed. There was a distinct softening of the bones of the calvarium in their entirety, especially on the right side, on account of infiltration with tumor tissue. The dura mater was quite adherent, and was the seat of extensive tumor thickening, which, over the right frontal region, had caused a marked depression in the region of the 2d and 3d frontal convolutions. The brain, the pituitary and pineal glands, the ears and sinuses, were normal.

The marrow of one of the lumbar vertebræ which was examined was pale, but presented larger and smaller areas which were soft and somewhat reddish in color. The lower diaphysis and the lower portion of the shaft of the right femur were hyperemic, the marrow being grumous. There were also a few small areas, about 3 mm. in length and breadth, which were whitish in color, and somewhat firm. The marrow of the ribs appeared very scanty, the bone having a hollowed out appearance.

The case briefly reviewed exhibits many noteworthy features. Perhaps the first in importance is the extensive distribution and abundance of the tumor-like foci; secondly, the noteworthy involvement of widely scattered tissues which, however, are closely related functionally; namely, the diffuse involvement of the marrow of one of the long bones, the vertebra and the calvarium, the extensive nodular infiltration of the serous surfaces of the pleura, peritoneum, and dura mater, and of the omentum, the lymph vessels of the mesentery, and the liver and spleen. The hematopoietic systems, marrow, liver and spleen, were accordingly extensively involved. In association with these lesions, the mediastinal, cervical, peripancreatic, portal, mesenteric and retroperitoneal lymph nodes were likewise extensively infiltrated. On the other hand, the organs not related to the lymphatic system, at least in the functional sense, such as the brain and lungs, were practically uninvolved.

It will occur to all that this case presents an analogy, at least anatomically, to the picture presented by leukemia and pseudoleukemia. The absence of metastases in the lungs and brain, the comparatively slight involvement of the other organs, with the exception of the suprarenals, and the extensive involvement of such widely separated but functionally closely related organs is at least very striking.

The malignancy of the tumor, judged by its tendency to invade the surrounding tissues, as in the lymph nodes, is not marked. Thus, only in a few places have the individual lymph nodes forming the large masses of glands, become confluent. In other words, the growth in the lymph nodes shows no marked tendency to break through its capsules. This, however, has occurred by local invasion from the iliac nodes into the testicle and retroperitoneal nodes.

The histology of the tumor nodules, which is essentially the same throughout the various tissues, may be described briefly.

The changes found in these bones, on account of their great interest and importance in connection with the case, may be described first.

The marrow of the femur presents a remarkably homogeneous appearance. The smaller and larger spaces between the bony lamellæ are filled with large spheroidal cells, which are markedly uniform in appearance. The picture presented is thus totally unlike that of normal red bone marrow. The cells are large and, when free, distinctly spheroidal in shape. The protoplasm is markedly eosinophilic, and, except with the high power, is homogeneous. The nuclei are large and round, usually distinctly vesicular, showing a more or less marked chromatin network, and one or more nucleoli. In the smaller cells the nuclei tend to stain more deeply and uniformly. A delicate stroma with rather wide meshes is occasionally made out, especially in the sections of the calvarium.

The cells greatly exceed in size the plasma cells. They possess no specific granulations characteristic of the myelocytes. These cells resemble strikingly those depicted by MacCallum, and possibly those shown by Quackenbos, in their cases of multiple myeloma. The sections of the calvarium and the body of one of the lumbar vertebræ present a similar histological picture to that of the femur. With MacCallum we may consider the cells as directly related to the myelocytes, and probably derived from, or similar to, the large non-granular forerunners of these cells.

The tumor nodules found elsewhere in the body show a striking similarity in their histological picture, especially in reference to the cells which we have described as replacing the normal cells of the marrow. They are more or less spheroidal in shape, and only when the cells are closely aggregated do they tend to become polygonal. The nucleus is usually central, and is relatively large, but occasionally it is eccentric as in the plasma cells. The chromatin content varies greatly, some of the nuclei staining intensely and diffusely, but usually the chromatin is scattered throughout the nucleus in the form of rods and strands, or forms a more or less definite network. The majority of the nuclei have distinct nucleoli which are large and take an intense red color with the methylgreen-pyronin stain. There is a distinct nuclear membrane. Numerous mitotic and karyolytic figures, and irregular divisional forms are seen, which may be held to be an evidence

of the rapid proliferation of the tumor cells. The cytoplasm stains rather intensely with eosin, and appears somewhat stippled or granular, and with methylgreen-pyronin stain has an intense reddish color. No granulations of any variety could be detected in sections stained with Giemsa or Unna's polychrome. Many of the nodules are surrounded by well-defined connective tissue capsules, from which are given off delicate septa which form a supporting reticulum for the tumor cells. No fibrin could be demonstrated between the cells with Weigert's stain.

In many places no connective tissue stroma is made out, and the cells are separated merely by a granular intracellular substance in which are nuclear fragments and red blood cells. The reticulum does not, therefore, appear to be a constant or predominating feature of the structure. The vascularity of the tumor tissue varies greatly, but nowhere is the presence of new formed blood vessels a striking feature. Areas of hemorrhage and necrosis are frequently encountered.

We wish to call your special attention to the peculiar and highly colored small nodules in the liver and spleen, which have imparted such a brilliant appearance to the slices of these organs. The color is a deep purple. The Kaiserling preparations here shown have practically preserved the color, although the tint is paler than in the original specimen. The largest of these nodules does not exceed 5 mm. in diameter. Sections of these nodules show the same tumor cells in abundance, mixed, however, with numerous red blood cells, with a fair number of nucleated red blood cells, resembling normoblasts. These nodules we consider approach closely the type of tumor which Ribbert describes as erythroblastoma. It is known that under certain pathological conditions, as in syphilis (Askanazy), the blood forming function of the liver may reappear in extrauterine life.

We have thus in our case a tumor-like process in which large and non-granular cells and erythroblastic cells are present. The presence of these nucleated blood cells or normoblasts may possibly be accounted for upon the basis that the tumor cells being the original and undifferentiated elements of the bone marrow or "Stammzellen" have developed into erythroblasts, a view which has been expressed by Scheele and Herxheimer.

The presence of red blood cells in a well preserved condition in small groups and in close association with the tumor cells is certainly striking. There is no evidence of the breaking down

of the red corpuscles, and no hematoidin crystals are present, which one would expect if an actual hemorrhage had occurred.

So much at this time for the essential structural features of the tumor. It was in the bone marrow from the femur and the vertebræ that the most complete replacement of normal tissue by the characteristic tumor cells was encountered, and in the sections under the microscope we can see that the spaces between the trabeculæ are entirely filled with large round cells, such as compose the tumor masses elsewhere. It is this extreme involvement of the bone marrow, such as is found in all the leukemias and the various types of myelomas, that has inclined us to the belief of the myelogenous origin of the tumor-like process.

The case before us exhibits in the present state of our knowledge, insuperable difficulties in the way of a satisfactory classification. The case in our opinion must be simply considered as one of myelosarcomatosis, since the tumor is distinctly myeloid in type. It will be remembered that no characteristic blood picture was presented during life. In certain places the sections, especially of the splenic vein, show large clusters of these tumor cells, but evidently these cells, at least as judged through two examinations of the blood, were not regularly circulating in the blood. According to Borst's classification, at least for the present, the diagnosis of myelosarcomatosis must be made. In view of this it may be wise to review briefly Borst's recent article upon the classification of the sarcomas.

According to Borst, the sarcomas arising from the lymphoid and myeloid tissues of the hemapoietic system, the so-called hematoblastic sarcomas, are of great interest, since they approach in type many diseases whose classification, in the present state of our knowledge, presents great obstacles. Recently he has emphasized the difficulties which exist in distinguishing the inflammatory growths from real tumor processes, and drawn attention to the existing tendency to classify the disease processes which anatomically resemble new growths with the infectious granulomas. According to Borst, any classification of these tumor processes must be considered a temporary makeshift. He divides the hematoblastic sarcomas into two broad groups.

In the first group belong the primary solitary tumors which are malignant. A variety of names has been given to such tumors: Lymphoma sarcomatosum; Lymphocytoblastosarcoma, or Sarcoma lymphaticum, lymphadenoides or lymphomatosum, or sim-

ply Lymphosarcoma, and, by Ribbert, Lymphocytoma. These tumors in their structure resemble lymphatic tissue in a more or less complete manner as to reticulum, and as to the lymphocytic character of the tumor cells.

The tumors vary among themselves as to the predominant character of the cells, in some cases the cells resembling the large cells found in the germinating centres of the lymph nodes, rather than the small lymphocytes. These tumors spread, as do the typical sarcomas, by means of the lymph or blood stream. Such sarcomas arise from various places and organs, especially the lymph nodes, spleen and marrow. Another tumor with the same characters in regard to its origin, growth, and manner of spreading, resembles in its structure myeloid tissue. These tumors arise from the marrow and possibly, according to Borst, from the periosteum, as primary solitary tumors, the so-called myelocytoblasto-sarcoma, or, briefly, myelosarcoma or myelocytoma.

In rare cases these tumors have even a closer resemblance to the parent tissue, by the formation of rudimentary blood-forming islets; that is, by the presence of hematoblastic cells, these tumors representing the so-called hematoblastic sarcomas of Hansemann, Albrecht, and Borst.

The second group of tumors, which stand in marked contrast to the tumors of the first group, is called by Borst primary multiple systemic sarcoma, or sarcoma-like new growths, which Sternberg has lately reviewed. In this large group the tumor process is confined to one portion, or to the whole, of the blood-forming system; that is, the myeloid or lymphatic system, the process appearing simultaneously in many widely separated places, or beginning in one place and then rapidly spreading to other places in the same kind of tissue, or in other tissues which are functionally similar. In this way a true metastatic process may be simulated. The difficulty in eliminating this factor is increased because of the presence in many organs of small microscopic lymphoid nodules which do not in the strict sense belong to the blood-forming system; namely, the lymphatic apparatus of the mucous membranes, liver, kidney and lung.

Sternberg and others have recently called attention to the normal occurrence of myelocytes in the spleen and lymph nodes. Furthermore, certain organs, especially the liver, spleen and kidneys, in their fetal life have blood-forming functions, red and white blood cells being manufactured in these situations, and

under certain circumstances this may occur in later extrauterine life. It is therefore not surprising that these organs, the liver, spleen and kidney, are affected in such systemic diseases. In other words, the lymphocytes, myelocytes and erythroblasts occurring in tumors may develop *in situ*, instead of being carried from a distant focus.

Sternberg and Borst are of the opinion that the true sarcomas should be separated from the leukemias and pseudoleukemias and the multiple myelomas. The myelomas show in their macroscopic appearance a striking similarity to the cells of the bone marrow from which they have been developed. In these affections there is thus missing the aberrancy from the normal type shown by the cells of the real sarcomas, a diagnostic feature which has been so much emphasized. The process is an example of a systemic tumor-like hyperplasia, unassociated with malignancy, local destruction and replacement of old tissue by new tissue, or with metastases. These tumor-like processes are made up either of myeloid tissue, in other words, a myelogenous leukemia, or of lymphatic tissue. The close relationship between the leukemias and the pseudoleukemias is at the present time recognized, as the latter can turn into the former, the difference being purely quantitative in respect to the changes in the blood picture presented in either disease. Thus, when the white blood cells formed in the marrow enter the blood system the disease is leukemia; when they do not it is pseudoleukemia.

The general pathological status of these so-called systemic diseases has not been positively determined. It is possible, according to Borst, that some may later be grouped with the infectious granulomas, in the same way that we now recognize the false pseudoleukemia; in other words, the diffuse lymph node tuberculosis. The other systemic diseases of this class which have not an infectious basis should be, according to Borst, classified as homotypic true blastomas, making them analogous to the benign connective tissue new growths, as an example, the so-called multiple fibromatosis of the nerves.

Opposed to these homotypic systemic tumor-like processes are the heterotypic malignant and infiltrating tumors which form metastases. These multiple primary systemic malignant tumors have been classified under the name of sarcomatosis. They have been separated into two groups, the melo- or lympho-sarcomatoses, according to Sternberg, on the basis of the tissue from

which they have originally sprung or which they resemble. In some of these tumors the atypical cells have entered the blood current. Such cases are referred to then as leuko- or chloro-leuko-sarcomatoses, or, better, as lympho- or chloro-sarcomatoses with a leukemic blood picture. When the cells possess granules, one speaks of a myelosarcomatosis. When the cells do not enter the blood current, one speaks simply of lymphosarcomatosis or of myelosarcomatosis. According to Borst, a tumor of the latter class has not yet been recognized, but it seems probable that it will be found.

Our case does not correspond to this type of Borst, inasmuch as the cells have no granules. Nevertheless, we feel justified in placing the case in this group of myelosarcomatosis, on account of the myelogenous character of the tumor cells. It is best not to call it a myeloma, since this name has been applied only to processes involving the bone marrow alone. Nevertheless, our case resembles in a great many respects a malignant myeloma, in other words, a myeloma which has formed metastases.

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Sternberg. Ergebnisse d. allg. Path., Lubarsch u. Ostertag. Neunter Jahrgang, II. Abt., 1903.

SUPERFICIAL INGUINAL HERNIA, WITH REPORT OF A CASE.*

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So much has been written concerning hernia that it would seem the subject had been exhausted, and had become devoid of any attractions. But the more unusual forms always excite interest and certainly merit a description, especially if, as in this instance one more case of superficial inguinal hernia is added to the comparatively small number reported. Undoubtedly more cases have been observed than one would be led to believe from the number one finds recorded in literature. For this reason it would be inaccurate to base a percentage of occurrence on the cases we find described and reported.

In a paper published in the *Annals of Surgery* (June, 1903), Coley records 937 cases of inguinal hernia, and of this number five cases are mentioned of the superficial inguinal variety. No individual operator with the exception of Macready, reports as many as does Coley, who, as stated, reports five. Erdmann, in his article on "Strangulated Hernia,"† reports three cases. If we take Coley's statistics we arrive at a frequency of occurrence as represented by one to about 187. This proportion based on the experience of a single operator will not, however, stand the test of accuracy, and will not represent the true relative frequency, as can be readily understood.

Superficial inguinal hernia was first described by Küster in 1887, and in his first communication he describes three cases. The reported cases as far as I am able to find them are to be credited as follows: Küster 3, Hulke 1, Moynihan 1, Macready 6, Wahl 1, Goebell 1, Moschcowitz 1, Fano 1, Curling 1, Erdmann 3, Coley 5, and Warran 1. The case of Warren is included in a recent

* Read before The Society of Alumni of Bellevue Hospital, New York, February 1, 1905.

† Medical Record, March 12, 1904.

Reprinted from The Medical News, March 18, 1905.

article on "Undescended Testicle," by Odiorne and Simmons. Moschcowitz, in an article published in the *Medical Record* of January 10, 1903, adds to his series another case. He credits it to Salzman, who found the condition on autopsy. Thus we find including the Salzman case a total of 26 cases, not including the case herein reported. To Moschcowitz is credited the collection of 17 cases, to which number are to be added those of Coley, Erdmann, Warren and the one of the author. The total number of cases then is 27.

This hernia may be considered as a congenital variety, not in the sense, necessarily that it exists from birth, but that the conditions leading to its production are most favorable. In other words, the cavity of the tunica vaginalis remains patent and communicates with the general peritoneal cavity. With this non-closure of the vaginal process and the testicle lying in an abnormal position it is readily understood how a hernia may protrude into the open and receptive pouch.

Macready, in his Treatise on Ruptures, classifies superficial inguinal hernia among the interstitial herniæ, and gives the following variations as regards these: (1) The viscera may be placed between the parietal peritoneum and the fascia lining the abdominal muscles; (2) between the internal and external oblique muscles; (3) on the surface of the external oblique, between it and the skin. The relations in superficial inguinal hernia are as given in the third variety. The essential features of this type, then, are a condition in which the hernia and testicle lie directly upon the aponeurosis of the external oblique and having a covering of only skin and superficial fascia. The infundibuliform fascia and the cremaster muscle are but little developed, which facts, perhaps, are in keeping with the etiology of the condition. A reference will be made to this point later on.

Eccles is authority for the statement that interstitial herniæ may be associated with imperfect or abnormal descent of the testicle. On the other hand, they may be found in males whose testicles have descended into the scrotum. Further, interstitial herniæ occur in females in a greater proportion than in males. The proportion, according to Langton's statistics, is as two to one. In 129 cases of interstitial hernia, observed during a period of twenty-four years, Macready found that some abnormality of the testes existed in 73 4/10 per cent., and in 67 1/10 per cent.

* Annals of Surgery, December, 1904.

the testis was completely retained or had only partially descended. What proportion of superficial inguinal hernia is associated with imperfect descent of the testis I am unable to state accurately, but I think it may be safely said that nearly all cases have some degree of cryptorchism.

There are at least five varieties of inguinal hernia associated with imperfect descent of the testicle—bubonocele, scrotal, interstitial, cruroscrotal, and superficial perineal (Eccles). Of all the conditions found associated with imperfect descent of the testicle, hernia is the most frequent complication. Perhaps 50 per cent. of the cases are thus complicated. Of the varieties as given above, bubonocele is the most frequent form of hernia that is encountered, and it is the form which would naturally be expected, for it is but an earlier stage of several other varieties.

In trying to discover a possible explanation of this condition, a great number of theories has been expressed by various authorities, and this very fact of divers opinion, shows that as yet, none has been satisfactory or really meets the conditions. Sufficient it is, to state in the scope of this paper, that more than likely an abnormal development of the scrotal portion of the gubernaculum has something to do with its production and must be considered a factor in the genesis. By reason of this deficiency or insufficiency of the gubernaculum the protruding mass escaping through the external ring and finding its way into the scrotum blocked, goes in the plane of least resistance, or in other words, dissects between the skin and the aponeurosis of the external oblique. In addition we must take into consideration that in many instances of interstitial hernia there is to be found an abnormal development or certain retrograde changes in the structures forming the abdominal parietes. The fact that interstitial hernia occurs so frequently in females who have borne a number of children and in whom the musculature has become deficient and flabby, attaches special importance to this point. In males, then, given a tardy development in the muscular structures of the abdomen and a non-descent of the testis complicating, superficial inguinal hernia has at least a partial solution.

As to the proper procedure with imperfectly descended testicle complicating hernia, again there is a diversity of opinion. The remarks on this point are confined to adult cases. It is generally held that an abnormally placed testicle has no function, at least as far as the production of spermatozoa is concerned. Some

there are who believe in the internal secretion of even an atrophied testicle and for that reason never remove it. Reports of single cases in which the testicle has been transplanted to a scrotal bed are to be found, and the results are seemingly satisfactory. On the other hand, more cases abound in which the testicle much atrophied has retracted after a time, to a position high in the scrotum, just outside of the external ring and even into the canal itself. In a review of these unfavorable cases, pain is a most predominant symptom. Some authorities are firm in the belief that if for any reason orchidopexy cannot be performed, as for example, by reason of a short cord, then the proper procedure is to replace the testicle into the abdominal cavity. This has the objection that the testicle may again find its way into the canal, with a recurrence of original conditions. Again, there are cases in which testicles placed in the scrotum by operative measures, have gone on to full development. Others again consider orchidectomy the proper procedure. In view of these conflicting observations, it is clear that no one plan can be formulated to apply to the treatment of all cases. In every case, peculiar and distinctive conditions obtain and operative measures must be employed as best suit the occasion. In cases of radical procedure on one side, there is the satisfaction of knowing that a single properly descended testicle of normal development will carry on the bodily functions. Given a much atrophied testicle, with a shortened cord, firmly adherent to the sac, there is, to my mind, but one treatment—orchidectomy. Further, it is to be remembered, that an undescended testicle after the age of puberty, atrophies more and more, is a constant source of pain and is liable to undergo inflammation and malignant change.

To Dr. Ladinski I am indebted for the privilege of reporting this case. During his service at Gouverneur Hospital the patient was admitted and referred to me for operation:

Case.—S. M., 27 years of age; was admitted to Gouverneur Hospital on the evening of September 20, 1904. He is married and the father of several children. Right scrotum has always been empty. Three months ago patient began to be troubled with "rupture" on right side. This "rupture" has been getting larger, but never had caused any great amount of trouble. It was always reducible. The undescended testicle at times caused inconvenience. At 4 P. M. on the date of admission, after some heavy lifting, the tumor suddenly became larger and he was unable to return it as before. The tumor increased in size, became more and more painful, and he began to vomit. At this time he was brought to the hospital. The case was reported to me as a strangulated femoral

hernia. The description of the House Surgeon was that a tumor about the size of a large fist occupies the region of the right external ring, somewhat above the usual location of an inguinal hernia and extends downward on the thigh to a point corresponding to the saphenous opening. The tumor is dense and the overlying tissues are very thin and somewhat movable. He gives the signs of strangulation. His temperature is 97° F. and pulse 80. An examination some thirty minutes later revealed a tumor as reported, and the man presenting signs of strangulation. The tumor extends in part below Poupart's ligament, but upward pressure shows the femoral canal free, and the protrusion really comes from above the ligament. The right scrotal sac is empty. An incision four inches in length above and parallel to Poupart's ligament was made. Skin and superficial fascia having been passed through, the tumor presented itself as lying above the external ring and resting directly upon the aponeurosis of the external oblique. Inspection revealed that it was composed of intestine and a small almond-sized structure, which upon later examination proved to be the testicle. The diverging pillars of the external oblique tightly encompassed the whole protrusion. The external oblique aponeurosis was the slit and the inguinal canal seemed entirely obliterated, that is, the internal and external rings were almost in a straight anteroposterior axis. The sac being opened the hernia revealed itself to be of the congenital variety. The point of stricture was found to be at about the site of what should be the internal ring and was due to a narrowing of the lumen of the peritoneal sac. The stricture was divided and the gut, which showed signs of gangrene, was treated with hot saline applications, and after about ten minutes, was deemed viable and was returned. Great difficulty was experienced in the attempt to separate the sac from the attenuated cord and atrophied testicle, and they were therefore sacrificed. The sac was ligated and returned to the abdomen. The steps of a Bassini operation were followed, with the exception that there was no cord to deal with. The patient reacted well from the operation and made an uneventful recovery, being discharged on the twenty-first day.

The special point of interest attaching to this case, aside from its being of the rare superficial inguinal type, is the fact that on first examination it had all the aspects of a femoral hernia. The first diagnosis as reported to me, it may be inferred, was made without the careful examination which should always be made of a large hernia in the inguinocrural region. The ability to displace the mass above or below Poupart's ligament will establish the diagnosis as either inguinal or femoral hernia as the case may be. Macready, moreover, has this to say regarding interstitial hernia: "The ventral swelling of interstitial hernia is not always confined to the area of the abdomen. When it becomes prominent it may fall over the thigh and simulate femoral hernia."

As to the diagnosis of the condition of superficial inguinal hernia, it is believed that it can be but speculative and not posi-

tive. This is to be said, however, that perhaps additional stress can be put on the possibility of the existence of such a hernia when there is a mal-position of the testicle complicating.

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MALAKOPLAKIA OF THE URINARY BLADDER—REPORT OF TWO CASES.

By A. M. Pappenheimer, M.D., *Pathologist.*

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The term "malakoplakia" was first used by von Hansemann[1] in 1903 to designate a curious disease of the urinary bladder, characterized by the presence in the mucosa of irregular, slightly raised, fungoid plaques of a yellowish color. The first cases of this affection were, however, described by Michaelis and Gutmann[2] in 1902, in an article entitled "Ueber Einschlüsse in Blasentumoren." Since then similar cases have been fully described by Landsteiner and Störk,[3] Gierke,[4] and Minelli[5]. In all ten previous cases have been recorded.

The disease appears to be one of later life, the ages of the subjects varying from forty to seventy years. The majority of the cases have been in females. Including the author's cases, eight have been found in women, three in men; in one case the sex was not stated. An associated pulmonary or urogenital tuberculosis has been found in five of the twelve cases. In one, the bladder itself showed tuberculous ulcerations, but no tubercle bacilli could be demonstrated in the plaques themselves. The number of cases is as yet too few to decide whether the coincident occurrence of tuberculous lesions in other viscera is more than fortuitous.

The plaques vary in number, size, and distribution. There may be but two or three, as in one of our cases, or there may be several hundred. In size, they vary from small nodules not larger than miliary tubercles to large, flat, confluent outgrowths several centimeters or more in diameter. They are rarely raised more than 1 or 2 mm. above the level of the mucosa. The edges are abrupt or slightly overhanging. The surface is flat; often in the larger plaques somewhat depressed or umbilicated in the centre. The color is grayish yellow. Most often, the plaques are set into relief by a vivid zone of hyperemia in the adjacent mucosa. This zone, probably because of post-mortem changes in the blood pigment, may appear brown or grayish.

[1] Reprinted from Proceedings of the New York Pathological Society, New Series, vol. vi., April and May, 1906, Nos. 3 and 4 (April meeting).

It is difficult to decide from the gross appearance whether the epithelium is continuous over the surface of the plaques. In several cases it has been stated that the epithelium was ulcerated over the centre of the plaque; subsequent microscopic examination has then shown it to be deficient over the entire outgrowth.

The distribution of the lesions is highly variable. Sometimes they are most closely aggregated about the fundus, sometimes about the trignoum; most often they are scattered over the entire mucous membrane. In but one case (Michaelis and Gutmann, Case III.) have they been found in the ureters and pelves.

There has been a remarkable uniformity in the descriptions of the histological appearances of these structures. Save for minor points, the authors are entirely in accord, and it is only in the interpretation of the lesions that their views diverge.

The plaques consist principally of large, oval or polyhedral cells, closely aggregated, with little or no supporting fibrous reticulum. The cells are limited to the mucosa and superficial portions of the submucosa. They are of varying size, but are as large at least as a liver cell. The nucleus is usually eccentric. It may be vesicular, but it is more commonly small and deeply stained. The cytoplasm is strongly oxyphilic, and often presents a foamy or vacuolated appearance. At the base and sides of the plaques there is often a considerable amount of infiltration with lymphoid cells. Small round cells are also scattered amongst the large cells in the plaques. The surface of the plaques is invariably denuded of epithelium, and no transition is to be observed between the epithelial cells at the margin of the plaque and the large, polygonal cells of which the plaque is composed. Blood vessels are quite abundant, and consist of thin-walled capillaries, most often directed vertically.

Emphasis has been laid by all writers upon certain hyaline, spherical bodies, wrongly termed "inclusions," since they occur both within and without the cells. These bodies vary from just visible granules or droplets to large, concentrically ringed structures, four or five times the size of a red blood corpuscle. They stain readily with aqueous hematoxylin, without previous mordanting, and give a distinct Berlin-blue iron reaction with potassium ferrocyanide and HCl. According to Gierke, they also give micro-chemical reactions for calcium. Their nature and significance have been the subject of considerable discussion. They are supposed to be derived from broken down red corpuscles directly

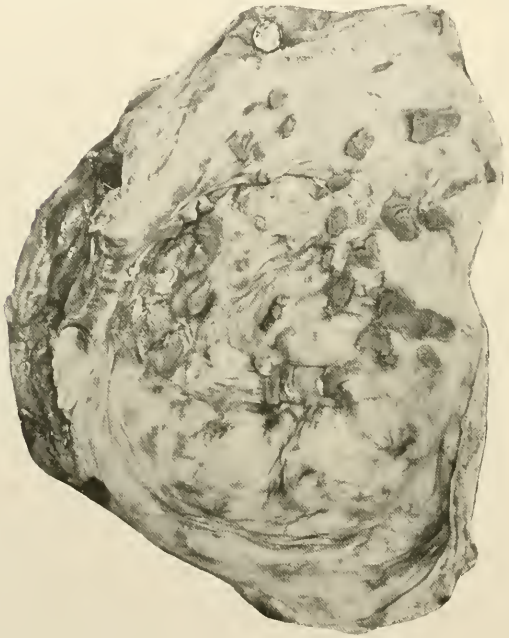
by Landsteiner and Störk,[3] or indirectly from indigested blood pigment by Minelli[5].

Bacteria have been found in the plaques and commented upon by all the writers except Michaelis and Gutmann. They are found in clumps or colonies, in the deepest portions of the plaques as well as in the more superficial. They are frequently intracellular. Morphologically they resemble the colon bacillus. With Gram-Weigert's stain they decolorize more or less readily. They are not acid fast. Cultures have never been made and the nature of the organism is quite unknown. By Landsteiner and Störk the bacteria are thought to be of etiological importance; by von Hansemann, Gierke, and Minelli, they are regarded merely as possible, and by no means specific, incitants of the disease.

Concerning the nature of the process, whether inflammatory or neoplastic, the final word has not been said. Gutmann and Michaels, basing their opinion upon the faulty observation that the large, polygonal or ovoid cells were derived from the epithelium, considered the lesion as a form of benign epithelial neoplasm. Their third case, in which the plaques extended to the ureters and pelves, they considered as a more malignant variety of the process. All the other writers, save von Hansemann, who does not commit himself to a definite opinion, consider the lesions as infectious granulomata of inflammatory origin. The precise origin of the characteristic large cells is still a subject for speculation. That they are of similar origin to the phagocytic cells seen in other inflammatory processes seems beyond question. Their large size may, as has been suggested by Minelli, be due to the imbibition of fluid from the urine.

Two cases of this infection have been found in Bellevue Hospital during the past month. The first case was in a woman, sixty-four years old, who died shortly after admission to the hospital. The autopsy was made forty-three hours after death. Her bladder showed a few greenish colored, freely movable, superficial nodules, varying in size from 2 to 4 mm. situated on the posterior surface near the fundus. The trigonum was free. There was a moderate general cystitis, the cavity of the bladder containing slimy; whitish muco-pus. Microscopical examination of these plaques showed a characteristic histological picture, with large cells, very numerous hyaline "inclusions" giving the iron reaction, and numerous colonies of bacilli, somewhat longer than the colon bacillus, but having the same distribution and tinctorial reaction as those hitherto described.

The second case was also in a woman, fifty-three years of age, with advanced pulmonary and intestinal tuberculosis. The autopsy was made four days after death. The bladder exhibited is taken from this case. The mucous membrane is studded everywhere with the plaques, abrupt, raised 1 or 2 mm. above the surface of the mucosa. The color is yellowish or yellowish green. About the plaques there is the usual zone of hyperemia. In the



PHOTOGRAPH OF INNER SURFACE OF BLADDER.

region of the trigonum the plaques are confluent, completely surrounding the ureteral orifice. The size of the individual plaques varies from 1 mm. up to 2 or 3 cm. They are most numerous on the trigonum and on the posterior wall. The surface of the plaques is covered with a small amount of greenish exudate, which can be scraped off. The bladder wall is not apparently

changed, and the mucous membrane is elsewhere entirely normal. Histologically, the characteristic structure is obscured, partly by post-mortem changes in the tissue, partly also by inflammatory infiltration and necrosis. The characteristic "inclusions" giving the iron reaction could not be demonstrated, nor were the bacteria, which have been present in all previous cases, to be found. Since, however, there is no other known bladder lesion presenting a similar macroscopic appearance, the lesion must for the present be classed as one of malakoplakia.

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GUNSHOT WOUNDS OF THE SPINAL CORD; A PLEA FOR EARLY MYELORRHAPHY, WITH REPORT OF A CASE OF BULLET WOUND THROUGH THE LIVER, SPINAL COLUMN, AND CORD; LAPAROTOMY, LAMINECTOMY, RECOVERY.*

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Introduction—The surgery of traumatic lesions of the spinal cord is at present in a very unsettled state. Operators are not unanimous in their opinions or treatment of these injuries. True, many are changing from the very conservative teaching of a few years ago to a more radical position, yet the radicals do not take the advanced ground in certain destructive lesions of the cord, I believe they should. It is primarily to advocate active interference in the worst cases of spinal injury that this paper is written. While it deals only with gunshot wounds of the cord, the argument, proofs, and conclusions are equally applicable to the severer forms of fracture, dislocation and traumatism.

If my interpretation of the essential facts of experiment and of the remarkable results obtained in a few cases is correct, there is good ground for the hope that surgery will be able in the future to benefit a greater number of those cases hitherto, almost unanimously, abandoned to a miserable existence and inevitable death within a few months.

Mortality—From the war records[1] La Gard concludes that "this class of injuries are the most fatal in war hospitals, and their gravity increases as the wound approaches the head." Otis[2] in the records of the Civil War "places the mortality at 70 per cent. in cervical, 63½ per cent. in the dorsal, and 45½ per cent. in the lumbar regions respectively. The fatality is increased by

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wounds of the neighboring viscera. With involvement of the spinal cord, the termination is sooner or later in death." Makin,[3] from his experiences in the Boer War, states "that these cases were proportionately the most fatal of any of the whole body to the wounded, who left the field of battle or the field hospital alive." Vincent[4] publishes records of thirty-three cases with a mortality of over 67 per cent. In forty-two of Prewett's[5] cases the mortality was 76½ in the cervical region, 66⅔ in the dorsal and 50 per cent. in the lumbar region—an average of 64 4/10 per cent.

From the forty-six cases which are here collected the total mortality in the cervical, dorsal and lumbar regions is 36 per cent., 55 per cent. and 66⅔ per cent. respectively; the average being 52½ per cent. This would seem to show that with modern aseptic treatment in civil practice the mortality is not nearly as high as in war time and preantiseptic days. When death occurs soon after the injury, it may be due to high situation of the wound, visceral complications, hemorrhage or septic infection. Death, after a longer period, is due almost always to a septic condition, proceeding from bed sores, multiple abscesses or a surgical kidney. Indeed, this last condition is very frequent.

Pathology—Gunshot wounds of the spinal column show all grades of severity from mere perforation of the spinal canal to complete division of the cord. These wounds are usually associated with a concomitant traumatic fracture of the vertebra that does not in itself demand special consideration, except for the results which may follow displaced fragments of bone pressing upon or perforating the cord. Frequently there is also present lesions of the abdominal or thoracic viscera or the vital structures in the neck.

External to the dura there may be compression by hemorrhage, depressed fragments of bone or the bullet. The dura itself may be perforated by the splinters of bone or by the bullet, both or singly. The cord may suffer from concussion or compression by extravasated blood, fragments of bone or the bullet. Multiple minute, or single large hemorrhages may have taken place into its substance or it may have been perforated, or completely severed by the bullet. The statistics regarding serious injury to the cord itself in Prewett's cases are as follows: Completely severed, 4 times; crushed, 6 times; piereed by bullet or splinter of bone, 6 times; compressed by bone, bullet or blood clot, 10 times.

In the cases included in this paper, these injuries occur in this proportion: Cord completely severed, 10 times; cord crushed, 4 times; cord pierced by bullet or splinter of bone, 5 times; cord compressed by bone, bullet or blood clot, 11 times.

In nine of Vincent's thirty-three cases the bullet was found within the spinal canal. In forty-two of Prewett's cases it was in the spinal canal thirteen times, and in these forty-six cases it occupied this position eleven times—*i. e.*, 24 per cent. Septic infection may be present and increase the gravity of the condition, "as all gunshot wounds are presumably septic from the infections carried by the bullet, particles of clothing or skin" (La Garde). The microscopical changes in the cord are those of destruction of tissue by the traumatism, and a degeneration of the various nerve fibres and cells above and below the site of the lesion.

Inflammatory changes will follow and end in the production of new connective tissue. If infection is present, the appearance varies from the mildest to the most severe grades of septic meningitis and myelitis. By consulting the histories presented, further details of the pathological changes may be obtained.

Symptoms—The spinal cord is a living cable composed of thousands of insulated filaments and their nerve cells for the transmission of motor and sensory impulses, and the production of various reflexes necessary to the preservation of life and health. It is also functionally divisible into segments corresponding to each pair of spinal nerves, and the various important reflexes have been accurately located within these segments. Furthermore, the relation of these spinal segments with reference to the spinal column is known.

The symptoms which follow cord injury are produced by an interference with the conductivity or interruption of the continuity of the nerve fibres, or a disturbance or destruction of the nerve cells reflected in various motor and sensory symptoms, and alteration in the reflexes in parts situated below the seat of the lesion. A complete epitome of the varying symptomatology will be obtained from a study of the cases here reported, and it will be unnecessary to enter into their consideration at greater length now, except to add one caution from Walton[6] "that we have no symptoms or group of symptoms which will prove to us within a reasonable time whether the cord is severed or not."

Treatment—What patients should be operated upon? I would answer that *all* who survive the initial shock of the injury to

the cord and its complications. By a study of the cases in literature it is very clearly proved that we cannot determine except within very wide limits the exact condition present within the spinal canal after a gunshot wound. Therefore, the damaged region should be laid bare in order that the extent of the injury may be ascertained and the proper treatment instituted.

In the forty-two cases I have selected as bona fide instances of gunshot wounds of the spinal cord among those reported by Prewett there is a total mortality of 71 per cent. in unoperated and 61 per cent. in operated cases. In the forty-six cases here collected the figures are 69¼ per cent. of mortality with noninterference and 42½ per cent. with operation. This improvement in the total results in the last ten years is striking, and is due in great measure, I think, to the operations being done earlier, quicker, and with aseptic technique. The lower mortality after operative treatment is a strong argument for such interference.

Conditions Demanding Operation—It is generally conceded that laminectomy is imperative in the following conditions: 1. To remove the bullet or spiculæ of bone, or particles of clothing; 2, to remove blood clots; 3, to arrest hemorrhage; 4, to allow oozing in traumatic œdema of the cord; 5, to prevent pressure and sepsis by free drainage; 6, in advancing paralysis.

The next indication, viz., 7, to suture a severed cord is a proposition not universally accepted. It is to this phase of the subject, however, that I wish to devote most of my time.

Various Views Regarding Operation—Bolton[7] would restrict operative influence in all cases "to the removal of foreign bodies, to facilitate disinfection, to prevent more extensive necrosis and to facilitate drainage." He believes "that total lesion of the cord is irremediable * * *, permanent, and requires no treatment." Lloyd[8] states that "it becomes therefore an impassable barrier to surgical progress when the cord has been definitely injured;" also, "it is the compression, and only the compression without injury to the cord, that can be benefited, and this may be taken as an axiom in surgery of the spine."

These opinions published previous to 1902 give a correct representation of the accepted surgical teaching at that time, and, I am sorry to say, is at present the almost universally accepted practice. To be sure, one now and then has apparently with little success protested against this plan of noninterference in the very serious cases, as Prewett, writing in 1898, asks (in these cases

with severed spinal cord), "shall we leave them to survive or perish without attempting to improve their chances of recovery? Would it not be better to do a laminectomy, remove all foreign bodies, spiculæ of bone, the bullet, if possible, and the devitalized tissue of the cord, thereby diminishing the dangers of meningitis and myelitis, and promoting more prompt healing of the wound?" And Walton in 1901 wrote that "owing to the decided limitations of our diagnostic ability, it is well to promptly resort to operation upon all patients except the moribund and those with great displacement, and give them the benefit of the doubt." Sachs[9] said: "It is still the fashion to delay surgical interference until everything else has been tried, whereas, if surgical interference was to do any good, it should be practised at once." Dana[10] voiced the old contention that "if by clinical observation one could be sure that the cord was crushed, one should not recommend operation." Starr says that "if there is evidence of a total transverse division of the spinal cord an operation is useless." Vincent,[11] writing in 1903, advocates active interference. He sets forth statistics that expectation admits an excessive mortality, on the contrary interference has saved life.

Prewett quotes the following case reported to him by Briggs, of St. Louis. This is not a case of bullet wound, but bears directly upon our contention:

A boy, aged 17, had his spine crushed by a falling tree, and symptoms of complete division of the cord followed. Laminectomy was performed on January 5, 1898, three weeks after the injury; removal of the posterior arches of the ninth, tenth and eleventh dorsal vertebræ. The cord had been completely severed at the level of the tenth dorsal vertebra. "I at once conceived the idea," writes Dr. Briggs, "of cutting off the ends of the cord and attempting to reunite them." Four sutures of fine catgut were passed transversely through the ends of the cord and membranes tied, bringing the severed ends of the cord into opposition. There was gradual improvement in the boy's condition, partial control of the bladder, motion of the muscles of the knee in one limb and in nearly the whole of the other limb.

Next follows the celebrated case of Stewart-Harte,[15] which ought to have revolutionized the teaching and practice in this class of injuries. Inasmuch as the record of this case is so important for my purpose, I shall quote it somewhat at length:

Case I.—Stewart-Harte.—Total severance of the spinal cord; suture; partial re-establishment of function. The writer states that: "Previous to this case there is no authenticated case on record of union with restoration of function of a completely transverse section of the spinal

cord in man." They evidently knew nothing of Estes' or Briggs' cases, just quoted, which in inception and accomplishment antedated theirs by several years.

A woman, aged 26 years, was shot twice with a .32 calibre revolver. One bullet was easily removed from beneath the skin in the right lumbar region. The other entered one inch to the right of the seventh dorsal spine, and passed directly into the spinal canal. There was complete abolition at once of motion and sensation below a line around the body at the level of the tenth dorsal spine. Above this area was a belt of hyperæsthesia reaching to the ensiform cartilage. Just before the operation this also became anæsthetic. Superficial and deep reflexes of the lower extremities abolished.

An operation was performed three hours after the injury under ether anæsthesia. The right lamina of the several dorsal vertebra was crushed in, the left fractured at its base. The spinous processes and laminæ of the seventh and eighth dorsal vertebra were removed. There was a rent in the dura through which the bullet was seen; this was enlarged and the bullet extracted, also some small splinters of bone. The cord had been completely severed, and the ends were three-fourths of an inch apart (proved by five witnesses). The ends of the cord were approximated by three chromicized catgut sutures. One suture passed anteroposteriorly through the whole thickness of the cord, and the other two transversely. The catgut frequently tore out before the ends of the cord were finally brought into contact. The dura could not be approximated. The wound was sutured over a gauze drain that was removed after twenty-four hours.

At the end of the fifth day, there were evidences of sensation in the lower extremities. The superficial reflexes returned on the seventh day, and the patellar on the twenty-first day. At the end of sixteen months the patient could voluntarily flex the toes, flex and extend the legs, flex and extend and rotate the thighs. She could slide out of bed into her chair by her own efforts, and stand with either hand on the back of a chair. The bowels were under perfect control. About one pint of urine was passed three times during twenty-four hours. She was sometimes incontinent during sleep. The menses were regular. She had sense of touch, temperature, pain and position all over. The muscles were moderately rigid, and there was present on both sides marked but easily exhausted ankle and patellar clonus. The deep reflexes of the tendo Achillis, ligamentum patellæ, and hamstring tendons were marked. The rectus abdominis reflex was seen on both sides. There were no reactions of degeneration and no trophic changes in the skin or nails. No bed sores developed.

Under date of April 9, 1906, Dr. Stewart, in a personal communication, says:

"The patient is now able to take a few steps with the aid of braces and crutches. Sensation remains about the same as at the time of the previous report."

The idea, however, back of this advice for early interference is not to attack a severed cord, primarily, but to operate in the

hope of finding and alleviating the less serious conditions, before compression of the cord has produced secondary degeneration. As this sets in at a very early date, operations for its prevention must be correspondingly early. The same proposition holds good at present.

The plea that I make is that the worst patients, namely, with complete division of the spinal cord, should be operated upon and sutured at the very earliest moment, and not be consigned by a policy of inaction to an inevitable death. I will here set forth the reasons, proved by practice and experiment, for such advice.

Reasons for Radical Operations—First as to the anatomical possibility of suturing several cords where there has been loss of substance.

a. The feasibility of suture of the spinal cord has been demonstrated in the living when there has been separation of the ends to the extent of three-quarter of an inch. (See the cases of Estes, Briggs, Stewart-Harte, and Fowler quoted later.)

b. In work on the cadaver I have demonstrated that coaptation is possible after resection of three-quarters of an inch in the dorsal and one-half of an inch in the cervical regions, such resection is followed by retraction of the ends of the cord to a much wider interval than represented by the segment removed.

The last I have demonstrated in spite of the statement of Chipault[20] that coaptation under such conditions is "anatomically impossible." There is, however, a very essential step in the operation, which renders coaptation after the loss of three-quarters of an inch possible, and this is full extension of the spine and head.

Experiment I.—I resected three-fourths of an inch of the spinal cord, in the lower dorsal region in a powerfully built male, of probably 55 years of age. The body had been frozen in the morgue, and later injected with an arsenic and formalin solution. As formalin hardens all tissues, some allowance must be made for this factor. With the trunk in flexion over a block, and the head flexed on the sternum, the ends of the cord retracted to over an inch. The three sutures, recommended for the operation and used by Stewart-Harte and Fowler, were passed without any difficulty by raising and separating the cut dura by traction sutures, as I suggest. With the trunk in the flexed position, coaptation could not be obtained of less than half an inch; but by placing blocks so as to extend the head, thighs, and trunk, the severed ends of the cord came together nicely. The effect was then noted of a change of position. The thighs were dropped to the table (semiflexion), no change; the head was allowed to drop in a flexed position, there was still apposition of the

ends; but as soon as the body was placed flat on the table, the ends of the cord began to pull away until contact was preserved only over the central parts of the fragments. Fixation of the spine then in a fully extended position I consider a very essential factor in maintaining coaptation until healing takes place.

I think, however, that in the living freer mobility of the cord is present than in the subjects prepared for dissection, and that cadaver work does not express the full possibilities of spinal cord resection and suture in the living. If this is the case, coaptation could be secured during life even after loss of an inch of the cord.

Experiment II.—The cadaver was that of a very old woman, ten days after death, not injected. The dorsal spine was flexed ventrally, and fixed in the bowed condition of old age by bony and fibrous changes. The cervical vertebræ above the sixth showed fair mobility. The spinal cord was exposed by removing the laminæ of the fifth cervical to the second dorsal vertebræ. The spinal canal and cord were markedly bowed forward. Extending the head lessened the tension on the dura, but did not change the curve in the spine. The dura was opened, and one-half inch of the cord resected opposite the seventh cervical vertebræ. The cord was loosened up by tearing off (with a hooked probe) two or three of the pointed attachments of the ligamentum denticulatum above and below the site of section. The usual sutures were placed in position, the head and trunk extended as fully as possible, the severed ends of the cord could not, however, be brought into contact. The presence of the deformity in the dorsocervical region prevented such a result. The interval was, however, reduced to about one-eighth of an inch. Division of the roots of two pairs of nerves on the lower portion of the cord did not allow of any gain in bridging the gap. The conclusion was plain that in this instance full extension of the spine being impossible, the ends of the cord could not be approximated.

Experiment III.—Cadaver of a male adult, strong and muscular, dead ten days, not injected. The dorso-cervical region was exposed and the same amount of cord resected opposite the vertebra prominens as in the preceding instance. Two pairs of serrations of the dental ligament, above and below the site of section, were torn away by a hooked probe, the usual three sutures passed, the body was now dropped to the flat table and the head alone extended. The ends of the cord came into neat adjustment. The head was allowed to drop to the table but the ends still remained in close apposition; but the cord above and below the sutured area drew out and was distinctly narrowed.

From this experiment we may conclude, that, with a normal spine, one-half inch may be resected from the spinal cord in the dorsocervical region and the ends coaptated by sutures. I think that division of at least two pairs of teeth of the dental ligament, at their point of attachment to the dura, and full extension of the

head are necessary in securing this result. The sutures did not show any tendency to tear out and held the cord well, even under considerable tension.

As to the manner in which the sutures should be inserted, I think the plan practised by Stewart-Harte and Fowler is the best. One is passed anteroposteriorly through the centre of the cord, the other transversely through the cord, one near its ventral, the other near its dorsal surface. To the objection that this method destroys some fibres and cells in the cord, I would reply that so does the method of passing sutures through the piaarachnoid, for no one can insert sutures into this delicate membrane, that will be broad and deep enough to hold anything, without catching up some of the cord substance. Furthermore, the first method coaptates the ends well, and will not tear out so readily as those passed through the piaarachnoid. Damage must inevitably be added to the severed cord by sutures however placed. The plan here favored, however, does as little damage as any; it holds the fragments in good contact, the sutures are much quicker and easily placed.

Second—As to the possibility of severed cords uniting—waiving for the moment the question of the regeneration of the nerve fibres.

a. In animals hemisections of the cord is always followed by union. This is true of complete section but in this instance there is the retraction of the ends, and the interval is filled in by a blood clot first, followed by a cicatrix.

b. In human beings Chipault[19] states that some cases of wound of the cord by a stab have been reported by Professor Raymond (1897), and by Huguier and Bernard (1900). Once, the stab had penetrated at the level of the seventh cervical process, another time, in the third and fourth dorsal. In the two cases there was complete recovery. Chipault adds that all observations agree that this is what ordinarily happens in this kind of lesion.

c. In gunshot and fracture dislocation lesions attended by destruction of cord tissue the several ends have been united in five cases with evidence of union in all.

Third—Is such macroscopical union attended with true microscopical union or regeneration?

a. Experimental evidence. Bearing directly upon this fundamental question of the regeneration of the nerve fibres and cells

in the spinal cord, as conclusively proved by the two cases just recited, are the following observations by Schäfer[16] that:

"Eichorst and Naunyn, after transection of the spinal cord, found nerve fibres traversing the cicatrix, which they considered traceable to an afferent root adjacent, being able to follow them to a spinal ganglion."

Under the chapter of regeneration of the spinal cord, Schäfer states:

"It must be remembered that some recovery of function is possible without regeneration of the tissues. In dogs regeneration of cells has been described, also the extension of nerve fibres across the scar without any regeneration of nerve cells; whilst by others neither regeneration of nerve cells nor extension of nerve fibres into the scar could be found. Karyomitosis has been found in the ependyma cells of the central canal at the site of lesion, but none in the nerve cells proper. The origin of the new nerve fibres that in some cases traverse the scar has not been ascertained."

Again from the following brief summary of the exhaustive paper by Strobe[17] bearing upon the possibility of the growth of spinal axones we learn that:

"The fibres of the posterior roots, at the level of the lesions, grow out from the side of the spinal ganglions, and push their way a distance between the fibroblasts of the cicatrix. These new fibres look like the regenerating fibres of divided peripheral nerves. These at forty-five days did not reach the axial plane of the cicatrix. From the cut end of the cord young fibres are even after two weeks growing into the scar tissue. A tongue-like bundle of such fibres was seen in the lateral (crossed pyramidal) column growing into the scar. Fibres wound along and around the new vessels. These fibres are, however, limited to the edges of the cicatrix, and in forty-five days did not fully traverse the cicatrix. These fibres are not of full size, they break up into numerous fibrillæ at their ends, produce many collaterals, but at forty-five days did not produce anything like genuine regeneration of the cord."

He speaks of areas of softening and cysts above and below the lesion and the dilatation of the central canal. No attempt was made to bring the ends of the severed cord together, but mention is made of blood clots that separated the ends of the severed cords after twenty-four to forty-eight hours.

b. Evidence furnished by the results of suture of severed cord in human beings. In a publication of recent issue Estes[14] says:

"I have done two complete and one partial exsection of the spinal cord. As a matter of interest, though it is not important at all, I should like to state that my first case of complete resection—I mean the resection of a section of the whole thickness of the cord—was done in *October, 1889.* I made a complete resection of a disintegrated cord, at the first lumbar vertebra, removed about three-quarters or an inch of the cord, squared

off the ends, and brought them together with catgut sutures and sutured the dura mater over the united cord. The operation was done on the tenth day after an injury from indirect violence (the man fell from a high telegraph pole). The cord was lacerated entirely through, and many fragments of the bone had been driven into the spinal canal. The man was considerably improved as regards the sensory and trophic disturbances, but never regained the use of his lower limbs. I again exsected about half the thickness of the cord in the lower dorsal region (ninth and tenth dorsal), which was lacerated and entirely disintegrated by sharp spiculæ of bone. I left the anterior columns only intact, then I drew the ends of the lateral and posterior columns together by catgut sutures, and sutured the dura over the united ends. Sensory and trophic paralysis improved almost immediately. The man finally recovered the use of his lower extremity, and the use of the flexors of the right extremity, so that with the aid of a steel and leather brace on the weak side he was able to walk with comparative ease. His sphincters entirely recovered."

A study of the experiments that have been conducted on the spinal cord in the past proves very conclusively that in no case was 1, the severed cord sutured; consequently 2, a blood clot formed between the ends of the cords, which retracted to a greater or less extent; however, 3, fibres (sensory) grew upward from the spinal ganglion into the cicatrix; and 4, fibres (motor) grew downward into the cicatrix; 5, these fibres did not re-establish the continuity of the spinal cord; but I ask, is it any wonder that they did not, inasmuch as the ends of the cord were separated by a blood clot—later cicatrix? They did the best they could in the short space of forty-five days. What might have happened in several months, or what have happened if the ends of the cord had been held in apposition by sutures?

A more positive proof of the possibility and actual development of new nerve fibres is obtained in the following history of a case with autopsy and post-mortem findings by Shirres[18]:

"Regeneration of the axones of the spinal neurones in man: Gowers refers to the possibility of regeneration taking place, and gives the results of pathological section of the spinal cord in a case that had shown symptoms approaching transverse division of the cord with a fair return of motion and sensation. In the crossed pyramidal tracts and other areas of the cord at the seat of the lesions were found a number of very fine axones in a situation where normally large nerve fibres are usually seen. He was of the opinion these smaller fibres were regenerated axones."

The following is the case cited by Shirres of complete severance of the spinal cord with a gap of one-half inch between the ends, in the service of Dr. Armstrong, in the Montreal General

Hospital, 1902, which deserves critical attention. No attempt was made to suture the divided ends at the time of the operation, as there was no hope entertained that the patient would survive. After months of treatment carried out by Dr. Shirres and his assistants "there was no return of the reflexes or alteration in the flaccid state of the limbs. But truly wonderful results were achieved as few trophic or vasomotor disturbances appeared in the extremities eleven months later."

Dr. Shirres and Dr. Armstrong after this interval decided to make an attempt to secure regeneration between the ends of the severed cord. Accordingly at a second operation, a segment three inches long of the spinal cord of a large dog was placed alongside of the severed ends of the patient's cord, the interval now between the ends having increased to one and one-half inches. No attempt was made to diminish this gap. A few fine sutures united the piaarachnoid of the one to the other, the dura was closed, wound sutured, and a plaster jacket applied; the patient made a perfect recovery from this unique operation. A month passed without change, but the electrical and massage treatment was carried out by Dr. Shirres and a corps of assistants as before.

"By the fifth week the patient was conscious of flatus in the lower quadrant of the abdomen. This he had never experienced before. Six days later he was conscious of the passage of the catheter, and two days later he informed the orderly his bowels were about to move, and could tell when fæces passed the rectum (anus?). On this date, he complained of sensations of pins and needles in the right foot, and a week later, of the same symptoms in the left. Two months after the operation, he had subjective disturbances in both feet as high as the knees. Little other change was noted until the eightieth day after the operation, when for the first time, with the plexímeter, a certain amount of tone was noticed in the muscles of the flexor surface of the thigh and leg."

This very interesting progress was terminated about a month later by the death of the patient from a large abscess of the right kidney, which gave no urinary evidences of its presence.

Examinations of sections of the cord showed the following condition:

"Above the site of the lesion there was typical ascending degeneration of the columns of Goll and Burdach, the direct cerebellar and Gower's tracts. Sections below the lesion showed definite degeneration in the direct and crossed pyramidal tracts. The dura and tissue at the site of the grafting experiment showed a mass of minute myelin sheaths of nerve fibre, which laid closely adherent to the dura, and when traced upward and downward, united with the segments of the cord above and below, demonstrating the fact that regeneration of the axones of the

spinal neurones had taken place to a limited extent. At the time of the operation, the dura between the two segments was perfectly clear of nerve fibres to the naked eye. Sections of the lower segment showed it to be in fairly healthy condition. In the cauda equina 90 per cent. of the nerve fibres were to all intents and purposes normal by the Pal-Weigert stain."

The importance of these post-mortem findings justifies, I think, this lengthy abstract of a very interesting history. Dr. Shirres states that he thinks the success in this case was largely due to the patient having been so assiduously treated by electricity, the prevention of contractions, and the frequent movement and massage of the extremities. Another case by Fowler (see Case XVI.) confirms the preceding results.

Name for the Operation—So far as I have been able to determine Estes was the first to use the terms myelorrectomy and myelorrhaphy for section and suture of the spinal cord. As the words are formed similar to words already in general use their adoption is recommended.

Conclusions as to Operating for Severed Cords—First—Chipault says in reference to stab wounds of the spinal cord followed by complete recovery " that this is what ordinarily happens in this kind of lesion."

Second—Experimental work on (animals) clearly demonstrates that sensory fibres have grown upward and motor fibres downward into the blood clot or cicatrix, filling the gap between the severad ends of the cord. This attempt at regeneration has been made under the most adverse conditions—viz., retraction of the severed ends of the cord, the presence of an intervening blood clot and insufficient time for recovery. If these unfavorable conditions were eliminated, what might happen?

Third—The results in the cases of Estes, Briggs, Stewart-Harte, Shirres, and Fowler[21] (see Case XVI.) in instances of complete severance of the cord, and of Pilcher,[22] Pegram,[23] and myself in perforations of the cord by the bullet show beyond any question that the spinal cord does regenerate.

Fourth—In every case of complete transverse lesion of the cord not operated upon, the patient died in less than a year. Of the five cases (Estes 2, Briggs 1, Stewart-Harte 1, Fowler 1) which were operated upon the ends of the cord brought together by sutures one patient died within ten days, the other patients were living and improving at the date of the various reports (one two and one-half years and one over four years since the injury).

In view of these facts, how can we escape the conclusion that all patients with completely divided spinal cords should be operated upon, and an attempt made to secure end to end union of the severed fragments, by properly placed sutures?

Laminectomy, Myelorrectomy, and Myelorrhaphy—How may this result be obtained? This question can be answered by briefly recounting the steps in the operation of laminectomy. A free median incision through the skin to the spinous processes, a clean severance of the tendons of the spinal muscles, and a rapid stripping up of the muscle mass so as to bare completely the laminæ of from three to five vertebræ. Pack the wound with hot gauze, compress for a few moments to check the free, mostly venous hemorrhage; then, with the muscles well retracted, you will have a dry field. With a rongeur forceps bite off the spinous process of one vertebra to its base and open into the spinal canal, gnaw away the laminæ laterally with rongeur or cutting forceps, sever one lamina after the other until the spinal canal is opened for the desired extent. Leave no sharp bony points. Osteoplastic methods take time and are not necessary as the spine is not appreciably weakened by this procedure. Deal with the conditions as you find them, with the minor details I will not delay you; open the dura for the full extent of the wound, place two to four traction sutures in its cut edge; they work much better than clamps or tenaculæ and are not in the way.

Remove the bullet if present, speculæ of bone, foreign bodies as clothing and blood clots, the latter preferably with a stream of saline solution. By drawing on the traction sutures and lifting up (backward) the dura, the cord is brought into better view. If it is completely severed treat it in a surgical way by carefully trimming off the bruised and lacerated ends (myelorrectomy), and then bringing them into coaptation by fine chromic catgut sutures (myelorrhaphy), introduced well away from the severed ends, so that in tieing them they will not cut their way through the soft cord. One suture is passed anteroposteriorly through the centre of the cord, the other two from side to side; one near the ventral, the other near the dorsal surface of the cord. To aid in the coaptation of the ends use the expedient of extension of the heads, thighs, and trunk, which I have found in work on the cadaver assists this object very materially. In the upper dorsal and cervical regions greater mobility of the ends of the cord may be obtained by tearing off with a blunt hook the at-

tachment to the dura of two pairs of the serrations of the dental ligaments above and below the site of the lesion.

Tie the sutures in the cord, smooth out the mushroomed ends of the cord* and over them adjust the dura closely by three interrupted sutures. Close the rest of the dural opening loosely, so as to allow of an escape of any excess of exudate or cerebrospinal fluid. Drain from the dura outward with strands of silkworm gut or strips of rubber tissue for twenty-four hours, suture the muscular mass with interrupted and the skin with continuous catgut. Apply a moderate dressing. Maintain the trunk fully extended until the operation has been completed and a plaster jacket has been applied. The after treatment will be referred to later.

I shall not discuss the treatment of the less serious conditions, as their character, and the steps in their treatment have been shown at the beginning of this section, but minor modifications of the major proceeding are here given in detail.

Time for the Operation—The next question is, when to operate.

Again experimental work and the results of cases actually observed indicate the time, and that is at the earliest possible moment after recovery from the initial shock of the injury, or after dealing with the serious complications of abdominal or thoracic lesions so often associated with spinal gunshot wounds. We may be compelled by their severity and the imperative demand for their prior treatment to neglect the spinal condition for a time, but wait no longer than necessary, because it has been shown that delay leads to most serious consequences from degenerative changes in the cord, as a result of the presence of a blood clot, the bullet, or infection.

The degeneration begins at once, proceeds with great rapidity, and the final results are largely governed by the effects of such pressure degeneration.

The effects of delay upon the cord are bad, not only when it is fully divided, but also when it has not been severed, but is compressed by the bullet, blood clot, or depressed fragments of bone. Such delay renders abortive, I believe, in many cases the efforts the surgeon makes to repair the damage even in the milder cases. Fowler attributes the poor result in his case, compared

* In a severed cord the ends "mushroom" from contraction of the pia arachnoid; after suture this tendency still exists, but can be counteracted in a measure by close application of the dura as suggested.

to the remarkable restoration in the Stewart-Harte's, to the delay of ten days, which was necessary before he could operate, to the short time of three hours intervening in theirs. Therefore, we must operate at the earliest possible moment in every case, in order to frustrate as far as possible their bad effects.

The After Treatment—Judging by the improvement obtained by Shirres in his practically hopeless case, the after treatment is a very important factor in determining the degree of final recovery.

Immediately after the operation the indication is rest to the spine. This is secured by the ventral or lateral decubitus, the body being maintained in the fully extended position (dorsal flexion) as previously suggested. As soon as healing has taken place in from two to three weeks, the jacket should be removed, the patient kept flat on a water bed, if available, and active electrical stimulation with massage and passive motion of the trunk and lower extremities be undertaken. Retention apparatus should be employed to prevent deformities in the limbs. The bladder must be regularly catheterized and irrigated under strict aseptic precautions, and the development of a surgical kidney prevented. Everything else pertaining to the preservation of strength, nutrition, and comfort must be utilized. As soon as possible get the patient out of bed and coax him to attempt to use his muscles. The stimulation to regeneration from voluntary efforts is very considerable.

Final Results—What results may we expect?

This is hard to say. It depends upon the site and extent of the cord lesion, the visceral complications, the previous condition, health, and age of the individual, the nature of the projectile, its size, velocity, and whether lodged in the canal or not, in the presence of hemorrhage within the cord, or within or external to the dura, whether the case is complicated by infection or not, and perhaps as vital as any factor the time that has intervened between the injury and the operation, and by the after treatment that is instituted and maintained for months.

With few exceptions, surgeons have in the past, when confronted by a case of complete disunion of the spinal cord, refrained from interference, and the death of the patient has inevitably followed in less than a year. The remarkable results obtained in these exceptions, furnish, to my mind, positive proof that with early myelorraphy, with careful technique, and with

persistent after treatment the outlook for this class of cases is not as hopeless as generally believed, in fact it is positively encouraging.

The patients that survive the initial shock and visceral complications, do have a chance; is it for us through delay and inaction to destroy that last chance? Therefore, I urge once more operation in every case of gunshot wound of the cord; operate as early as possible, do all that is necessary, even to suturing a severed cord, and carry out an after treatment that only terminates with the death of the patient or his ability to look after himself.

In closing, I wish to thank Dr. J. J. Moorhead for his valuable assistance in looking up the literature of this subject, and Dr. R. T. Bang for his help in translation from the French of matter bearing on the matter.

Case II.—Haynes and Moorhead. Gunshot wound of the liver, spine and spinal cord. Laparotomy-laminectomy. Recovery. V. C., twelve years of age, Italian girl. Brought to the hospital by an ambulance on October 28, 1905, in the service of Dr. Haubold. The child was accidently shot by a 32-calibre bullet, the wound of entrance being at the left of the median line, three and one-half inches below the ensiform appendix. The bullet could be felt and seen immediately at the right of the middle line of the back, at about the eleventh dorsal spine, where it lay just beneath the skin.

Examination—Pulse small. Extreme pallor; restless and very thirsty; dyspnœa; skin cold. Abdominal pain and rigidity. Patient complains of weight and tingling in lower extremities, which are very hyperæsthetic from the middle of the thighs downward. She screams with pain on the legs being touched, and cannot even bear the weight of a sheet. She moves her legs slowly and with difficulty, viz., flexes and extends legs at knee; very slight, if any, ankle motion.

Operation by Dr. Moorhead, assistant visiting surgeon. The wound of entrance was exposed by a vertical incision, and a wound through the left lobe of the liver disclosed, from which considerable blood had escaped. The liver wound was one and one-half inches from the anterior margin. The orifices of entrance and exit in the liver were closed by No. 2 chromic gut. There was no other visceral wound. The abdomen was made clean, a cigarette drain left in place, and the abdominal wound sutured. The bullet was removed from just beneath the skin, in the back, as previously indicated. On October 31st the abdominal drain was removed. The patient was still extremely hyperæsthetic over the lower extremities. There was incontinence of urine and fæces noted for the first time.

November 3d—Patient shows areas of anæsthesia corresponding to the terminal distribution of the musculocutaneous, small sciatic and external saphenous nerves, and motor and sensory paralyses of the

anterior tibial, posterior tibial and its branches, also of the anus and vesical sphincter. There is anæsthesia of the skin of the buttocks, perinæum, and inner side of the thighs, with the hyperæsthesia of the remaining anterior, posterior and lateral regions of the thighs. The condition is practically symmetrical. In view of the fact that the area of motor and sensory paralysis had extended since the receipt of the injury and a hæmorrhage into the spinal canal being entertained as the most probable cause, an exploratory operation on the cord was deemed advisable. Accordingly a laminectomy was performed on November 3d, by the writer, assisted by Dr. Moorehead and the house staff. Exposure of the bullet tract showed that it had come from between the laminæ of the eleventh and twelfth dorsal vertebræ, close to the base of the spinous process of the upper one. Both laminæ were slightly notched by the bullet. The laminæ of the eleventh and twelfth dorsal and first lumbar vertebræ were removed. No blood clot external to the dura was found. The dura showed a small round hole in its centre at about the middle of the field, and was incised for a distance of two and one-half inches. There was no blood clot about the cord, but it showed directly in its centre a small mushy area where it had been traversed by the bullet. The cord was not severed. There was no subdural hæmorrhage, hence the dura was sutured with a fine catgut continuous stuture. The deep muscles were sutured with heavier interrupted catgut. A few silkwormgut strands were left in place for drainage, and catgut was used for the skin wound. The course of the bullet into the spinal canal was through the intervertebral substance between the eleventh and twelfth dorsal vertebræ, through the centre of the spinal cord and the dura, then between the posterior arches as described, and into the muscles of the back. The bullet was not upset at all, and only showed some fine striæ made, I think, by its course between the laminæ of the vertebræ.

November 8th—The silkworm drain was removed. The wound closed and healed per primam. The course of the case was very tedious. There was incontinence of urine and fæces, and almost constant complaint of pain in the legs below the knees. There was a free discharge of bile from the anterior wound.

December 13th—The patient was seen by Dr. Leszynsky, the consulting neurologist to the hospital. At that date there was a slight general wasting of both legs, absence of both knee jerks and plantar reflexes. Fair irritability was present in the left peroneal nerve; questioned in the right. The galvanic current was used every other day, and right-angled splints applied to the feet.

By December 15th the patient had improved very much. Her appetite was good and the hyperæsthesia less. Patient can flex, extend, abduct, adduct, and rotate the thighs, she can extend and flex both knees, and can move slightly the tibialis anticus and the flexors and extensors of her toes. She can sit up.

December 17th—Anterior wound healed. Patient has regained control of bladder and rectum.

January 4th—Patient has been out of bed for a week; is practising standing.

Temperature and Pulse—At the time of the first operation her temperature was 104° and pulse 130. At the time of the second, the temperature was 100° and the pulse 120. Both reached normal about the second week, and with slight variations remained so.

I wish to call attention, 1, to the extremely unique wound of the spinal cord, which was traversed exactly through its centre by a 32 calibre bullet, at about opposite the intervetebral disc between the eleventh and twelfth dorsal vertebræ, and evidently traversing the fourth lumbar segment of the cord; 2, to the change from extreme hyperæsthesia in the legs to anæsthesia, and then the gradual regaining of the sensations in the skin to as far as the feet themselves; 3, to the primary muscular paralysis which had been gradually diminishing in extent; and 4, to the rectal and vesical incontinence for six weeks, with gradual regaining of control of these functions.

Present State—January 23, 1906—Examination by Dr. Hartwell, at Dr. Haynes' request. All sensation above level of crest of ilium is normal. Anteriorly, internally and externally sensations are normal as far as the knees. In the outer side of the right knee is a small area where pain and temperature sense are disturbed, while tactile sense is normal. On the surface of the right foot, extending from the base of the great toe on inner side, outward to base of third toe, from there upward to mid-ankle region, outward over external malleolus, and then across sole of heel, and from there up inner border of foot to starting point on the dorsal surface there is almost complete anæsthesia. There is no disturbance of sensation definitely made out in left foot. Posteriorly, there is marked disturbance of sensation for pain and for temperature, from the upper part of buttocks to just above the knee. That is about the distribution of the lesser sciatic nerve. Tactile sensation is little disturbed over this area. No disturbance in muscular sensation can be determined except in toes of the right foot. Muscular power for flexion at the hip is good, extension is good at the hip and the knee, but flexion of knee is very weak. Flexion of right ankle is absent, and extension poor. The left ankle is weak for both. Reflexes at knee, at ankle and at sole are abolished, Babinsky sign absent. Patient, on standing with eyes closed, rocks from side to side markedly; with eyes open and hand touching some object, can stand steadily. She walks with some help, with typical ataxic gait. She describes her difficulty in walking as due, not to weakness, but "to not knowing just where things are."

February 9th—Both thighs react entirely normally, anteriorly and posteriorly. The right leg acts tardily to both faradic and galvanic currents, but there is no reversal of the poles to the latter. The left leg shows the same condition in the posterior group of muscles, while the anterior tibial group shows lack of response to both faradism and galvanism, thus indicating a rather complete degeneration. The general

motor power is very much improved since January 23d, but the right anterior tibial group is still paralyzed. The reflexes and sensations are practically the same as on that date.

May 2, 1906—Examination and report of her present condition by Dr. William M. Leszynsky, neurologist of the hospital. The patient is now able to walk with slight assistance, or by the use of a cane. There is a mild degree of static ataxia; the gait is unsteady, with some dragging of the right lower extremity; the posterior thigh muscles are paretic on both sides, but more pronounced on the right side; partial foot drop on the right side due to paralysis of the peroneal muscles; loss of muscular sense in the toes of the right foot; there is an area of anæsthesia over the right leg corresponding with the fifth lumbar segment; the knee jerks, plantar and Achilles reflexes are absent; the faradic irritability is preserved in the peroneal nerves, but there is quantitative diminution in the reaction of the paretic muscles. The lumber muscles, iliopsoas and the quadriceps groups are intact. The bladder and rectum functionate normally.

Case III.—Haynes. Gunshot wound of the cauda equina. P. M., aged thirty-four, Italian; was shot at close range on December 15, 1905. The wound of entrance was three-fourths inch to the right of and on a level with the spine of the first sacral vertebræ. The wound was dressed antiseptically. At first the abdomen seemed somewhat sensitive and slightly rigid, and the possibility of a wound to some abdominal viscus from behind was entertained. The abdominal symptoms disappeared, but at this time the temperature began to rise until on the 17th it reached 104.6°, but the pulse was only 96. Slight stiffness was noticed in the muscles of the back of the neck on the second day after admission; by the following day there was so much contraction of these muscles as to draw the head backward and prevent the patient from lying on his back. The spine was slightly tender to forcible pressure. There was no paralytic symptoms except that there was retention of urine and incontinence of fæces.

With the subsidence of the abdominal symptoms, with the high temperature and rigid muscles of the neck I concluded that there was probably an infection of the spinal meninges, and that free drainage was required. Without any expectation, therefore, of finding the bullet, and merely to drain the spinal canal, a laminectomy of the fourth lumbar and first and second sacral vertebræ was performed on December 17th. The tract of the bullet led to the spinal canal. Just outside the laminæ a patch of cloth about three-eights of an inch in diameter was found. On opening the canal free hæmorrhage from around the dura followed. There was a wound through the dura. After splitting up the dura a free discharge of bloody serum (cerebro-spinal fluid) took place. The bullet was discovered imbedded exactly in the centre of the mass of nerve cords and removed. The wound within and external to the dura was drained by an iodoform wick, and the muscle mass loosely sutured and the skin partially closed.

From December 19th to 31st the temperature gradually dropped from 103° to 99½°, and the pulse from 120 to 90. On December 26th the

patient had a spasm of the facial (chiefly oral) muscles, with slight jerking of the head backward. No other spasm was noted. The wound discharged pus freely. The rigidity of the cervical muscles gradually disappeared.

On December 31st the temperature went up to 105 2-5° and the pulse to 128, without apparent change in his general condition. This condition gradually subsided, and in two weeks the normal was regained for morning records, with evening rises of 100° for temperature and 100 in pulse rate.

January 3d—The wound was granulated nicely, with considerable discharge of a thin, bloody, watery fluid.

Present Condition—January 31st—Still retention of urine and incontinence of fæces, but the incontinence of fæces is not so troublesome as at first. He has some sensation of a pending movement that gives him time enough to secure the bed pan or to go to the closet, but no control over the movement.

The wound has healed except for two very small sinuses at either end of the incision (which closed later). Patient is out of bed and around the ward. There is no rigidity of muscles in neck or back. The appetite is good, and pulse and temperature are running close to the normal.

A fairly complete record of gunshot wounds of the spine has been made since the paper of Prewett in *Annals of Surgery*, xxviii., 1898, and is here appended. Taking the statistics of Prewett's cases and those collected by Dr. Moorhead and myself, we have a representative set of cases embracing the so-called antiseptic era since 1879. These cases tabulated show the following statistics:

PREWETT'S CASES, 1879 TO 1896.

Case.	Condition of Cord.	Region.				Operation.		No Operation.		Remarks.
		Cervical.	Dorsal.	Lumbar.	Sacral.	Recovered.	Died.	Recovered.	Died.	
1	Disintegrated	1	..	..	..	..	..	..	1	Lived 23 days.
2	Degeneration	..	1	..	..	..	1	..	..	Lived 6 weeks, secondary meningitis.
3	Pierced by splinter of bone	..	1	..	..	..	1	..	..	Bullet in canal, pneumonia.
4	Compression	1	..	..	..	..	1	..	..	
5	Penetrated by bullet	1	..	..	..	..	..	..	1	Lived 42 hours.
6	Crushed, laceration	1	..	..	..	..	..	..	1	Lived 58 hours.
7	Compression	..	1	..	..	..	..	..	1	Lived 19 days.
8	Destroyed	..	..	1	..	..	1	..	..	Bullet passed through kidney.
9	Penetrated by bullet	..	1	..	..	..	1	..	..	Lived 54 days, septicæmia.
10	?	..	1	..	..	..	..	1	..	Walked, ataxic gait.

Case.	Condition of Cord.	Region.				Operation.		No Operation.		Remarks.
		Cervical.	Dorsal.	Lumbar.	Sacral.	Recovered.	Died.	Recovered.	Died.	
11	Lacerated	1	..	..	..	..	..	..	1	Lived 43 hours.
12	Laceration	1	..	..	..	..	1	..	..	
13	Hæmorrhage ?	1	..	..	..	..	..	1	..	Recovered.
14	Degenerated, softened	..	1	..	..	..	..	..	1	Lived 113 days.
15	?	1	..	..	..	..	..	1	..	Partial recovery.
16	Compressed	..	1	..	..	..	1	..	..	Lived 4 months.
17	Severed	..	1	..	..	..	1	..	..	Lived 6 days.
18	Perforated	..	1	..	..	..	..	..	1	Lived 17 days.
19	Crushed	..	1	..	..	..	..	..	1	Bullet in canal.
20	Bruised	1	..	..	..	..	..	..	0	
21	Nearly severed	..	1	..	..	..	..	1	..	Lived active life 5 years.
22	Uncertain	1	..	..	..	..	1	..	..	Lived 12 hours.
23	Cauda equina matted.	..	..	1	..	..	..	1	..	Lived active life 18 years.
24	Compression	..	1	..	..	1	..	..	..	Partial recovery.
25	?	1	..	..	..	1	..	..	..	Bullet removed, complete recovery.
26	Compression	..	1	..	..	1	..	..	..	Little improvement.
27	?	..	1	..	..	..	1	..	..	Died from shock of operation.
28	?	..	1	..	..	..	..	1	..	Partial.
29	Compression	..	1	..	..	..	..	1	..	Complete.
30	Not lacerated	1	..	..	..	..	..	..	1	Bullet in canal, 5 months alive.
31	Completely severed	..	1	..	..	..	..	..	1	Lived 3 months, bullet in canal.
32	Hæmorrhage	1	..	..	..	..	1	..	..	Lived 8 months before operation and 5 days after it.
33	Destroyed	1	..	..	..	..	..	..	1	Bullet in canal, lived 5 days.
34	Uninjured	1	..	..	..	..	..	..	1	Septic meningitis.
35	Nearly severed	..	1	..	..	..	..	..	1	Lived 24 hours, high dorsal.
36	Dura pierced	1	..	..	..	1	..	..	..	Complete recovery.
37	Split and contused	..	1	..	..	1	..	..	..	Bullet in canal, partial recovery.
38	Perforated	..	1	..	..	..	..	..	1	Lived 14 days.
39	?	..	1	..	..	1	..	..	..	Bullet in canal, complete recovery.
40	Compressed	..	1	..	..	..	..	..	1	Pleura, liver and kidney pierced by bullet, which lay in canal.
41	?	1	..	..	..	..	..	..	1	Lived 1 year, legs paralyzed.
42	Cord pierced	..	1	..	..	1	..	..	..	Partial, bullet passed through stomach.
	Totals	17	23	2	..	7	11	7	17	

STATISTICS FROM FORTY-TWO OF PREWETT'S CASES, COLLECTED FROM 1879 TO 1896.

Bullet found in the spinal canal, 13 times	31	per cent.
Cord severed ..... 4 times		
Cord crushed ..... 6 "		
Cord pierced by spiculæ of bone or bullet ..... 6 "		
Compressed ..... 10 "		
Cervical region, 17 cases	40+	per cent.
Operated, 6; recovered, 2; died, 4	66⅔	"
Not operated, 11; recovered, 2; died, 9	82	"
Total mortality	76½	"
Dorsal region, 23 cases	55	"
Operated, 11; recovered, 5; died, 6	56½	"
Not operated, 12; recovered, 4; died, 8	66⅔	"
Total mortality	61	"
Lumbar region, 2 cases	05	"
Operated, 1; recovered, 0; died, 1	100	"
Not operated, 1; recovered, 1; died, 0	00	"
Total mortality	50	"
Total mortality in operated cases	61	"
Total mortality in non-operated cases	71	"
Proportion of recovery in favor of operating	10	"

CASES COLLECTED BY DR. HAYES AND DR. MOORHEAD.

Cases.	Condition of Cord.	Region.				Operation.		No Operation.		Remarks.
		Cervical.	Dorsal.	Lumbar.	Sacral.	Recovered.	Died.	Recovered.	Died.	
1	Severed completely	..	1	..	..	1	..	..	..	Remarkable recovery (1).
2	Pierced	..	1	..	..	1	..	..	..	Almost perfect.
3	Cauda equina	..	..	..	1	1	..	..	..	Nearly complete (2).
4	Appeared normal	..	..	1	..	1	..	..	..	
5	Severed, crushed	..	1	..	..	..	1	..	..	Lived 8 days (3).
6	Crushed	..	1	..	..	..	1	..	..	Lived 3 months (4).
7	Compressed, soft	..	1	..	..	..	1	..	..	Shot through stomach, pyæmia, lived 11 months.
8	Completely severed	..	1	..	..	..	..	..	1	Lived 263 days.
9	Adherent to dura; cord not changed.	..	1	..	..	1	..	..	..	Shot through lung and liver; operation 3 years after injury.
10	Compression	..	1	..	..	1	..	..	..	Operation 4 years after injury; good recovery.
11	Pulpified	..	1	..	..	..	1	..	..	Lived 21 days.
12	Nearly sound	..	1	..	..	..	1	..	..	Lived 31 days.
13	Perforated by spiculæ	1	..	..	..	..	1	..	..	Lived 7 days.
14	Pressure	1	..	..	..	1	..	..	..	Resumed work in cotton mills.
15	Appeared normal clot in centre	..	1	..	..	..	1	..	..	Lived 6 days.
16	Completely severed	..	1	..	..	1	..	..	..	Fair recovery (5).
17	Bullet in canal, penetrated by spiculæ of bone	1	..	..	..	1	..	..	..	Fair recovery.
18	Destroyed	..	1	..	..	..	..	..	1	Mauser.
19	Destroyed	..	1	..	..	..	..	..	1	
20	Cord not touched	..	1	..	..	..	..	..	1	Martini-Henry.
21	Cord not touched	..	1	..	..	..	..	..	1	
22	Perforated near centre	1	..	..	..	1	..	..	..	Good recovery.
23	Appeared normal compressed by bone	..	1	..	..	1	..	..	..	Almost perfect recovery.
24	Clot in canal	1	..	..	..	..	1	..	..	Died from pneumonia.
25	Pressure from bullet	..	1	..	..	1	..	..	..	Practically full recovery (6).
20	Lacerated blood clot	..	..	1	..	..	..	..	1	Wound through liver, aorta, spine, lived 33 days.
27	Laceration one side	..	1	..	..	1	..	..	..	Fully recovered (7).
28	Perforated by bullet	..	1	..	..	1	..	..	..	Spleen and stomach perforated, laparotomy and laminectomy; fine recovery.
29	Contusion	..	1	..	..	1	..	..	..	Good recovery.
30	Severed completely	..	1	..	..	..	..	..	1	Lived 4 months (8).
31	Nearly severed	..	..	1	..	..	1	..	..	
32	?	1	..	..	..	1	..	..	..	Good recovery (9).

Cases.	Condition of Cord.	Region.				Operation.		No Operation.		Remarks.
		Cervical.	Dorsal.	Lumbar.	Sacral.	Recovered.	Died.	Recovered.	Died.	
33	Compression.....	1	..	..	..	..	..	1	..	
34	?	1	..	..	..	..	..	1	..	
35	?	..	1	..	..	..	..	1	..	
36	Entirely severed	..	1	..	..	..	1	..	..	
37	?	..	1	..	..	..	..	..	1	
38	?	..	1	..	..	..	..	1	..	
39	Compressed.....	..	1	..	..	..	..	..	1	
40	Severed.....	..	1	..	..	1	..	..	..	
41	Exposed.....	1	..	..	..	1	..	..	..	Almost complete recovery (10).
42	Suppuration.....	1	..	..	..	..	1	..	..	
43	Completely severed...	..	1	..	..	..	1	..	..	
44	Completely severed...	1	..	..	..	..	1	..	..	
45	Compressed by bullet.	..	1	..	..	1	..	..	..	Left hospital well (11).
46	Not determined.....	..	1	..	..	..	1	..	..	
	Total.....	11	31	3	1	19	14	4	9	

STATISTICS FROM THE 46 CASES COLLECTED, COVERING THE PERIOD FROM 1896 TO 1906.

Bullet found in the spinal canal.....	11 times	24 per cent.
Cord severed	10 "	
Cord crushed	4 "	
Cord pierced by spiculae of bone or bullet.....	5 "	
Compressed	11 "	

	MORTALITY per cent.
Cervical region, 11 cases.....	24
Operated, 9; recovered, 5; died, 4.....	44 4/9 "
Not operated, 2; recovered, 2; died, 0.....	0 "
Total mortality	36 4/11 "
Dorsal region, 31 cases.....	67½+ "
Operated, 21; recovered, 12; died, 9.....	42 6/7 "
Not operated, 10; recovered, 2; died, 8.....	80 "
Total mortality	55 "
Lumbar region, 3 cases.....	06⅔ "
Operated, 2; recovered, 1; died, 1.....	50 "
Not operated, 1; recovered, 0; died, 1.....	100 "
Total mortality	66⅔ "
Sacral region, 1 case.....	02+ "
Operated, 1; recovered, 1; died, 0.....	100 "
Not operated, 0; recovered, 0; died, 0.....	... "
Total mortality in operated cases.....	42¼ "
Total mortality in non-operated cases.....	69¼ "
Proportion of recovery in favor of operating.....	26¾ "

Case IV.—Vincent. Reported in *Revue de Chirurgie*, 1892. Abstracted in *Medical Magazine*, Vol. I., p. 89, p. 523. Man, thirty-one years old, wounded by a revolver, fracture of the first lumbar certebræ, partial paralysis of lower limbs, paralysis of bladder and rectum. Operation next day, fragments of bone and pieces of lead removed, no injury to cord, considerable immediate improvement of the paralysis, receding with the development of a myelitis which lasted one week, but rapidly returning afterwards. In one month he walked with crutches, and in two without support. The catheter was unnecessary in three weeks, the wound healed primarily.

Case V.—Vincent. *Ibidem*. Man, eighteen years of age, shot with revolver, fracutre of lamina of tenth dorsal vertebra, complete sensory and motor paralysis below. Operation on third day, entire ball found in spinal canal, also a fragment of bone, both removed. Very slight improvement the following day. On the sixth day patient was removed from hospital, left without use of catheter, and died two days later. Autopsy showed a crush of the cord through its entire thickness, with softening and suppuration.

Case VI.—Vincent. *Ibidem*. Man, thirty-seven years of age, shot with revolver, fracture of laminæ of eleventh dorsal vertebra, complete paralysis below. Operation on third day, ball and a large bony fragment removed from the vertebral canal. The wound healed well, and there was considerable improvement from the third day after the operation, but symptoms of myelitis were evident in three weeks, and the patient succumbed three months after the accident.

Case VII.—Kramer, reported in *Journal of the American Medical Association*, XXXV., 1900, p. 352. Woman shot with a 38-calibre revolver, bullet entered abdomen two inches below and three-fourths of an inch to left of ensiform, vomited blood, immediate and total paralysis below level of wound, great pain in upper half of body, purulent cystitis developed, bowels moved only by enema, extensive bed sores formed over all bony prominences below wound, motor paralysis of lower extremities with spastic contraction of flexors of legs and abductors of thighs, sensory paralysis to level of ninth dorsal spine, patellar reflexes absent. Operation performed on July 21, 1896, posterior arches of the eighth, ninth, tenth dorsal vertebræ removed, dura was found uninjured, and opened, the cord was soft and atrophic, and the bullet found in the body of the ninth dorsal vertebra, its point projecting into the spinal canal with two speculæ of bone, which were removed. The dura was not closed, muscles and skin sutured interruptedly about silkworm gut drain. There was primary union, and some amelioration of symptoms, but patient died on November 24, from pyæmia.

Case VIII.—G. A. Peters. Gunshot wound of the spinal cord, reported in *British Medical Journal*, October, 1897, p. 961. Woman, thirty-one years old, June 12, 1896, shot with 32-calibre revolver, the bullet entering the left side of the spine, near the eighth dorsal vertebra, went through laminæ and lodged in the body of the ninth dorsal vertebra. No operation done, as cord was thought to have been divided and condi-

tion hopeless. Died on 203d day after injury. Cord completely severed, and ends retracted for a distance of one and one-half inches.

Case IX.—Joseph Sailer. Bullet wound of spinal cord, reported in *Journal of Nervous and Mental Disease,* June, 1901. Man, shot on November 2, 1896, through lung, liver, and spinal cord, entrance of bullet one cm. to left of right nipple, exit two and one-half cm. to left of spine of twelfth dorsal vertebra, no suppuration, no hæmorrhage, complete paraplegia, loss of sensation in lower half of body, intense pain in back, followed by atrophy of legs, bed sores over all bony points, severe paroxysmal pain in thighs and lower portion of abdomen. Seven months after injury sensation began to return in left side, fourteen months after injury sensation normal in left limb, abolished in right leg to level of great trochanter, above this intense hyperæsthesia to level of the twelfth dorsal vertebra, complete abolition of rectal and vesical sensibility, motion and reflexes lost in right lower limb, while motion in left leg consisted of ability to flex thigh on abdomen, no motion below knee, knee jerk exaggerated, ankle clonus present. In June, 1899, a renal calculus was removed. In November, 1899, the patient began to suffer with severe fulgurating pains in thighs and lower abdomen. An operation was performed by Dr. Wharton, arches of the tenth, eleventh and twelfth dorsal and first lumbar vertebræ removed; cord exposed for four inches, dura greatly thickened, few adhesions to pia, which were broken up, no change could be seen in cord, wound healing perfect. This operation relieved pains absolutely, and some improvement followed, until there was anæsthesia and paralysis only of right leg and lower part of right thigh and total anæsthesia of left foot, movement at both hips and left knee joint, slight movement of left foot and toes, total anæsthesia over sacral area, and hyperæsthesia of perineal region, sexual reflexes only partially impaired.

Case X.—Lloyd, "Present Status of Spinal Surgery," in *Journal of the American Medical Association,* April and May, 1901. Male, shot four years (August 9, 1897) previously by a 32-calibre revolver in mid-dorsal region, a little to the left of the spine, immediate paraplegia and anæsthesia following, unconscious most of the time for four days, vesicorectal paralysis present, but improved after a few weeks. At the end of a year the patient could move his right leg, and had some sensibility in both. There was gradual improvement during past three years, general health being good. On examination the organs were found normal, bed sores on buttocks and ankles, ankle clonus exaggerated, also patellar reflexes, and gluteal spasm by pressure on sciatic nerves, feet in plantar flexion. On September 1st a laminectomy was performed, sixth to tenth dorsal vertebræ being removed evidences of fracture of seventh, eighth and ninth laminæ were found, pressure on cord existed over entire area, and evidences of old pachymenigitis. Recovery. Condition on leaving the hospital showed that reflexes were normal. The patient has recovered a great deal of power in lower extremities, although he still has to use crutches, but has no trouble with bladder or rectum.

Case XI.—Lloyd. (Case 166.) Weller and Van Hook, 1891, *American Journal of Medical Science,* CIII., p. 395. Wound from revolver

bullet in the third dorsal region, complete paralysis of lower limbs, removal of third dorsal arch, cord found reduced to a pulp, death followed twenty-one days later.

Case XII.—(Case 167, Lloyd.) Conley, 1891. Revolver wound in fifth dorsal region, complete paralysis of lower limbs, retention of urine and fæces, fifth dorsal arch removed, cord nearly sound, bullet found in back of vertebra, iodoform drain, but no union because of escape of cerebro-spinal fluid. Death thirty-one days later.

Case XIII.—Eskridge and Rogers, *Journal of Nervous and Mental Disease*, XXX., pp. 129-143, March, 1903. An alcoholic gambler, aged thirty-one, was wounded on February 23, 1898, by a 38-calibre revolver, the bullet entering left of front of middle line of neck, no wound of exit. There were no cerebral symptoms, but complete paralysis of left arm and leg, although patient could move right arm and leg. Operation was performed two days later, bullet had passed through fossa between fourth and fifth cervical transverse processes, fifth left cervical lamina splintered, it then entered spinal canal through body of fifth cervical. The bullet was extracted through back of neck, one inch below surface. Two pieces of bone (three-sixteenths and one-eighth inch respectively) pierced the cord proper, and three-eighths of an inch piece had entered membranes, causing much bleeding. The bullet had not affected the cord and latter was damaged by the spicule only. Death followed on seventh day. Autopsy showed softening about injured site of cord, some to right side and medullaward. Wounds due to the spicule, which did not penetrate more than half way toward central canal.

Case XIV.—Eskridge and Rogers (2). Male, aged nineteen years, shot on March 6, 1898, from behind, at a distance of fifteen to twenty feet by a 22-calibre Colt rifle. The entrance of the bullet was on level with seventh cervical spinous process. The patient was not unconscious, had no cerebral signs, left leg weak, but by effort can move both. Complete paralysis of left wrist and fingers, lessened left elbow power, while right side was normal, peculiar distribution of tactile sensation, pain and heat and cold sensation on left and right arm, and left half body, the deep reflexes were normal. Upon operation (apparently soon after entrance) the bullet and a "small spicula" of bone were found in the spinal canal at cervicocostal junction, external to membranes, but pressing on left lateral half of cord. The membranes were intact, little bleeding. Author mentions no other details. Patient was able to walk with a cane on May 1st. July 1st he had recovered except for defective muscular sense in left arm and leg, which he has to watch in walking. "Fall of 1898 * * * left town * * * resumed work in cotton mills * * *." As nothing was heard from him in response to letters, improvement was assumed.

Case XV.—Langdon and Wolfstein. Gunshot wound of the spine. *American Medicine*, December 14, 1901. Man, fifty-seven, was on April 21, 1901, shot by 32-calibre revolver in upper dorsal region, one inch to right median line on level with upper angle of scapula, no wound of exit. Examination sixteen hours after injury showed after he had recovered from shock, which was marked on entrance to hospital, that

paralysis of lower extremities was absolute, while touch of pain abolished below nipple line. No band of hyperæsthesia, retention of urine and fæces, knee jerk and ankle clonus absent. Plantar reflexes on both sides present, and flexor in direction. Operation seventeen hours after injury. Spinal canal was opened from third to sixth dorsal laminæ inclusive, showing a ragged hole through dura, slightly to left of median line, dura reflected, cord exposed for two inches, appeared normal. Bullet found and removed from body of fifth dorsal vertebra to left of median line. On April 23, plantar reflexes were present. On April 27 patient died.

Autopsy Record—Cord removed, no solution of continuity. On section a central clot was found occupying area of gray matter, cord in the region of the wound showed very extensive changes, fibres in white matter adjacent showed destructive vacuolization.

Case XVI.—Fowler. Suture of the spinal cord. Completely severed by a bullet. *Annals of Surgery*, October, 1905, p. 507. A. E., male, eighteen years old, was shot on April 28, 1903, in the back at a distance of thirty feet by a 32-calibre bullet. The wound of entrance was one and one-quarter inches to the right of the median line and on a level between the tenth and eleventh dorsal spines. Motor and sensory paralysis below a line around the body one inch above the iliac crest and midway between the umbilicus and symphysis. The bladder and rectum were paralyzed.

Upon operation on May 9th, the laminæ of tenth, eleventh and twelfth dorsal vertebræ were removed. The bullet was found lying transversely between the ends of the severed cord, imbedded in a large blood clot. The clot and bullet were removed, the ends of the cord approximated without difficulty by three fine chromatized catgut sutres, the dura being included in the sutures. The wound was drained by narrow strips of oiled silk and closed up, with this exception.

The patient was treated by massage, electricity and walking exercises in a cage-like support resting on wheels, somewhat like a "baby tender." With this support and braces to stiffen the knees, he could move about the hospital. During the remainder of 1904 and 1905, his bladder and rectal sensations improved. He can now tell when a movement of the bowels is imminent, and, at times, is able to retain it for a while. The same is true of the bladder. Both legs are spastic and quite useless for locomotion, except with the help of the apparatus mentioned. Sensation is practically abolished in the entire affected region, with the exception of an area five inches in length extending down the outer side of the right thigh, where some sensation is present. He cannot distinguish between heat and cold. Tactile sensations are recognized, but are usually referred to a point two or three inches distant from the point touched.

Case XVII.—Murphy. Report of pistol shot wound in neck. *New York Medical Journal*, LVIII., p. 414, August 29, 1903. Male, thirty-three years old, shot December 24, 1902, by 32-calibre bullet in neck, two and one-half inches above right clavicle, lodging in the skin above the scapula (presumably the right, although not so stated). Patient had motor paralysis of right leg and arm, no other data given. Following

week paralysis more marked (location not stated), and was taken home at end of four weeks. He suffered from bed sores, motor paralysis, right arm and leg, and right ulnar pain. Operation twenty-nine days after the bullet had been extracted (course not stated), laminectomy of fifth and sixth cervical, one-half inch spicula bone penetrated cord, site unstated, and depth not given, only benefit of operation was relief of pain.

A foot note (in small type, following a † mark) says: "Improved, walks with a cane, regained use of right leg, and partial use of right arm, bowels and bladder and bed sores normal * * *." "Time promises even better results."

Cases XVIII., XIX., XX. and XXI.—G. L. Cheatle. Four cases of gunshot injury to spinal column. *Journal of the Royal Army Medical Corps*, I., pp. 271-276, October, 1903. Below cases went to autopsy at Boer war. 1. Mauser wounded at 800 yards, eleventh dorsal wound, death on sixteenth day. No operation, destruction of cord. 2. Fifth dorsal, death twenty-four hours. No operation, membranes perforated and cord destroyed. 3. Martini-Henry, wounded ten yards' range. No operation, through seventh rib and seventh dorsal vertebra, and in latter leaden particles, cord and meninges untouched, death on seventeenth day. 4. Martini-Henry, wounded tenth dorsal. No operation, death on eighteenth day. Vertebral canal not entered.

Case XXII.—Pilcher and Onuf. Gunshot wound of cervical portion of spinal cord. *Annals of Surgery*, XXXVIII., 1903, p. 812. July 16, 1901, boy, eleven years old, shot by 22-calibre rifle, wound of entrance in the middle line in front, just below the episteral notch, motor and sensory paralysis from the clavicle down, no sign of injury to trachea, œsophagus, or any other structures in front of the spine, urination and defæcation involuntary, priapism present, respirations entirely abdominal. For the first ten days the tendency to pulmonary œdema and hypostatic pneumonia required constant treatment, involuntary defæcation and urination continued, but at the end of this time he regained control over the sphincter ani. At the end of three weeks he had regained partial and, ultimately, complete control of his bladder. On the fifth day first moved the toes of his left foot voluntarily, at the end of the fourth week he could move his left limbs normally, and had a good grip in left hand, improvement in the right side not so marked. On the seventeenth day he began to move his right leg, and at the end of the fourth week, he could flex the right leg on the thigh, but the movements were very weak. On August 12th the laminæ on the right side of the fourth, fifth and sixth cervicle vertebra were removed, but without locating the bullet, which was subsequently shown by an X-ray to lie in the spinous process of the sixth cervicle vertebra, no apparent destruction of the cord or membranes. The opening in the meninges was sutured, and the external wound closed over a small iodoform gauze drain. Recovery was uneventful, and the patient went home in two weeks.

One year after the injury he was examined by Dr. Onuf, who says that the most extension lesion was situated at the level of the eighth cervical to the second dorsal segments of the cord. The parts affected at this level were both anterolateral columns, probably chiefly the lateral

one, furthermore, both anterior horns, particularly the right, and probably the entire right posterior column. The nature of the lesion is difficult to judge, probably it was partly direct destruction by the bruising force, partly destruction by hæmorrhage, as is especially suggested for the anterior horn.

Case XXIII.—Marsh. Gunshot wound of spine. *American Journal of Medical Sciences*, 1905, p. 875. Male, twenty-five years of age, June 14, 1903, shot just below and internal to angle of left scapula by 32-calibre revolver, paralysis of legs was complete for two weeks, bowels and bladder also paralyzed, severe pain around lower part of thorax; after two weeks patient gradually began to recover use of his legs, right more than left, he can stand and move around a very little. Radiograph showed bullet lodged apparently in spine on a level with body of eighth dorsal vertebra, and a little to left of spinal canal. Six weeks later left pupil became slightly larger than right, sensation to touch and pain in legs delayed, especially in right; heat sensation normal, except upon right leg, when it is somewhat confused, analgesia present on outer side of right leg, ankle clonus on both sides; weakness of flexor muscles of both legs, most marked on left, transverse area of slight anæsthesia at level of twelfth dorsal vertebra and first lumbar vertebra, slight Babinski and patellar clonus on both sides, most marked on right, plantar reflex absent, marked diminution of power in left leg. Achillis jerk present on both sides. Upon operation the spinal cord was exposed from seventh to ninth dorsal vertebræ, inclusive, and appeared normal, the bullet had not entered canal, but had driven the posterior arch of the eighth dorsal vertebra inward, until it pressed on cord. Result, March 26, 1904, motions of spine normal, patient says he feels as well as he ever did, the left great toe has tendency to catch on floor, when walking barefooted, but gait is about perfect, knee jerks exaggerated, but muscular power of legs is normal, moderate degree of ankle clonus.

Case XXIV.—Miller. Gunshot wound of fifth cervical vertebra. *North American Journal of Homœopathy*, XXVIII., p. 509, August, 1903. Burly negro, 38-calibre bullet entered "below right ear * * * and lodged in outer one-third left clavicle * * *." Patient had no cerebral signs, "motor and sensory paralysis below clavicle and upper one-third of arm complete," retention of urine and fæces, he was able to eat. Operation forty-eight hours later, bullet wound "through laminæ of fifth cervical vertebra near spinous process * * *," pea-sized clot, cord normal. He died on third day. Autopsy revealed normal cord (no microscopical examination), hypostatic pneumonia, and death apparently due thereto, no sepsis.

Case XXV.—Alvarez. (Personal communication.) The patient was a farmer, aged about forty-five, who was seen and operated upon in June, 1904, about six months after he had been shot from behind by a 32-calibre bullet, entering from above right shoulder blade and passing downward to lodge at the costovertebral junction of the eleventh rib. He had also been shot in two other parts of body by two other bullets, but the wounds due to same had healed and had no bearing on his

spinal condition. Radiograph (by Caldwell) showed the bullet with its point in the spinal canal and this location was confirmed by laminectomy.

Symptoms—Patient walked with assistance; sensory paralysis to some extent below waist. "Numbness complained of below navel." Incontinence and constipation, but latter overcome by cathartics. No trophic changes; some atrophy in thigh and legs, but mainly of disuse.

Operation by Dr. Alvarez—Resection of eleventh rib and laminectomy of articulating vertebra. Bullet impinged upon cord, the symptoms being from pressure rather than from destruction of tissue.

Results—When last heard from (three months ago) the patient had practically recovered full function. He is "able to ride horseback * * * and walk without aid * * *." Dr. Pearce Bailey saw this case, as did Dr. Tilden Brown; former reports case in his recent *Diseases of Nervous System Resulting from Accident and Injury*, and improperly states (p. 217) that the missile penetrated abdomen and that the bullet lodged in the "fourth lumbar vertebra," he also credits the operation to Dr. W. T. Bull, who never even saw the case! (This statement is made by request of Dr. Alvarez.)

Case XXVI.—Poulton. Gunshot wound in liver, aorta and spine. *Australasian Medical Gazette*, July, 1904, p. 338. Patient was shot by .22 calibre Winchester rifle in February, 1904, had to journey forty miles by train, when examined at the hospital there was no nausea, no vomiting, no blood per rectum, paraplegia, retention of urine, no priapism, no abdominal distention, liver dullness, well defined, sensation in skin of left hip, but not over the rest of the lower extremities. Four days later, under a general convulsive attack, his legs moved, thereafter he could move feet a little, return of sensation in skin of legs, which was hypæsthetic in places. Post-mortem showed perforation of liver, aorta and first lumbar vertebra, the bullet was found in cauda equina, the termination of the cord was lacerated, and the spinal canal contained blood clot.

Case XXVII.—Fort. Laminectomy with report of a case, *International Journal of Surgery*, February, 1906. June 14, 1905, man, aged twenty-five, was shot by a .32 calibre revolver, and paralysis was immediate. The wound was at a point directly over the eleventh dorsal vertebra. Twenty hours after injury the following conditions were found: Motor paralysis complete from the wound downward. Sensory paralysis from the umbilicus anteriorly and the wound posteriorly to the knee. There is hyperæsthesia of the legs and feet, girdle pains, absence of patellar reflexes, and ankle clonus. Retention of urine and priapism.

Laminectomy was performed. The ball had fractured the spinous process of the eleventh dorsal vertebra and the right lamina, driving the fractured portions of the lamina into the spinal canal. The bullet had split, one third entering the spinal canal, while the remaining two-thirds had penetrated the soft parts, ranging inward and to the right. The portion of the bullet in the spinal canal had penetrated the dura and a small portion of the right side of the cord. This portion of the bullet was removed, and the dura and the rest of the field of operation were cleansed with saline solution. The wound of the dura was left open.

The rest of the wound was closed with interrupted silkworm gut sutures. He voided three ounces of urine on June 16th, but did not regain complete control of the bladder until June 19th. There was considerable escape of cerebro-spinal fluid. The motor and sensory paralysis of the lower extremities continued to improve, but hyperæsthesia below the knees persisted, although less marked. By July 15th the hyperæsthesia had disappeared, except immediately below the right knee. July 13th, the motor and sensory functions being practically normal, although he had not been allowed to walk. He has now fully recovered, except some pain on the external aspect of the right knee.

Case XXVIII.—Fagram. Bullet wound of abdomen, spleen, stomach, vertebra and spinal cord. *Annals of Surgery*, July, 1905, p. 83. July 11, 1904, woman, aged twenty, was shot at close range with a .32-calibre revolver. The bullet entered the body in the left axillary line over the eighth rib; abdomen tender on palpation with marked rigidity without dullness in the flanks. Complete motor paralysis of both lower extremities, though slight movement of the thighs, probably through the psoas muscles. Both lower extremities were hyperæsthetic, even the weight of the bed clothes being painful, with the exception of an area of the left leg supplied by the fourth lumbar nerve, which was anæsthetic.

She was operated upon and the bullet found to have passed through the lower border of the spleen, the mesocolon (cutting a branch of the splenic artery), in and out of the posterior surface of the greater curvature of the stomach, but no opening in the vertebra could be felt. The bleeding vessel was ligatured, the openings into the stomach closed, the spleen not bleeding and left alone, the abdomen closed about an iodoform gauze drain.

The patient reacted well from the operation. On the second day she complained of numbness in the legs, and could move the toes. On the fifth day she was incontinent of the urine for the first time. On the sixth day a bed sore developed over the sacrum, and the bowels moved involuntarily. The abdominal drain was removed on the seventh day, and considerable pus escaped.

On the tenth day an anteroposterior skiagraph showed the bullet to be in line with the body of the first lumbar vertebra, pointing downward. On the sixteenth day, a second X-ray from the side located the bullet apparently in the spinal canal. The ability to move the legs was increasing, but the pain in them was extreme. On the nineteenth day the spinal canal was opened, and the bullet found under the lamina of the twelfth dorsal vertebra in the right posterior quadrant of the spinal cord, which had been traversed by the bullet and was considerably lacerated. The bullet was removed, the rent in the meninges not closed, but drained by a wick of plain gauze, and the rest of the parts sutured in position around the drain. The patient was placed on a Bradford frame, face downward, recovery from the ether was good. On the second day after the laminectomy (twenty-first) voluntary control of the tibiales antici was present but weak. She moved the right quadriceps and adductors, and the left adductors. She was allowed to go home on August 31st, wearing a plaster jacket.

In December she could walk a few steps alone. She has regained almost complete control over the bladder, the bowels usually required an enema. On February 8, 1905, Dr. G. L. Shattuck examined patient.

There is a saddle-shaped area of anæsthesia to tactile and temperature sense, that is quite symmetrical. Starting on the sacrum behind and curving onward toward the trochanters and then turning downward and involving the posterior surface of the thighs as far as the upper border of the popliteal spaces, this anæsthetic area then extends to the inner side of thighs, including the labiæ. The whole surface of the left foot, including the ankle and a narrow strip on the right sole, are anæsthetic. Elsewhere, on both extremities, with the exception of the anterior surface of both thighs as low as the tibial tubercles, sensation was delayed though present. All muscles of both lower extremities give the reaction of degeneration. Though sensation was delayed, or altogether lost in places and normal in others, there were no sharp lines of demarcation to correspond with the distribution of any one nerve. A transverse myelitis must have been set up by the injury to the cord caused by the passage through it of the bullet, in order to produce the paralysis of the bladder sphincter, noted for the first time on the fifth day after the injury. The recovery from the lesion is necessarily a slow one, but if the patient continues to regain her lost functions at the present rate of progress, I believe we may look for a disappearance of almost all motor and sensory disturbances.

Case XXIX.—Vincent. *L'Etat actual de la chirurgie nerveuse.* I., 1903, p. 513, Chipault. Revolver bullet shot from a distance of two metres. Point of entrance on a level with eleventh dorsal, and a little to the left of the median line. Absolute paraplegia. Diminished sensation in the lower muscles and up to the umbilicus. Half erection. Resection of the posterior arch of the tenth and eleventh dorsal by means of chisel and mallet. Contusion to the left of the spinal axis, the meninges of which were not lacerated. The bullet was not recovered. During the days that followed, partial return of motion, then vesical contractions. The conditions improved, little by little, and at the end of several months the wounded man walked quite well, with a good vesical function.

Case XXX.—Ostermeyer. Gunshot wound in spinal cord. *Ibidem.* II., 1903, p. 516. A lesion of the spinal cord from a gunshot wound. A young girl of seventeen years was struck by a revolver bullet, shot from behind, on February 2, 1902. The point of entrance was found at the top of the tenth dorsal vertebra, five centimetres to the left of the median line. Complete paresis of the lower extremities with paresis of the bladder. Complete analgesia and anæsthesia. At the end of February, incontinence of urine. Many bed sores. Death occurred on June 11, 1892. The missile was found at the top of the eleventh dorsal in the canal of which it had completely severed the nervous contents.

Case XXXI.—Djemil Pacha. *Ibidem,* p. 654. A man, thirty-one years old, shot by a revolver in the lumbar region. Operation twelve hours after the accident. The spinal marrow was almost completely severed. The ball, lodged in the body of the twelfth lumbar vertebra, was removed. The patient expired some weeks after from exhaustion.

Case XXXII.—Maughinas. Gunshot wound of the spine. *Ibidem*, p. 690. Man, twenty years old, shot by a revolver in the right side of the nape of the neck, on a level with the fourth cervical vertebra. The patient had fallen unconscious from the shot, but, ten minutes later, he returned to consciousness. For six months after he experienced tingling along the right upper lower extremities. On the lower extremity the patient also experienced tingling, beginning at the knee and running down into the foot. There persisted during quite a length of time, a left torticollis which appeared after the injury. All these phenomena disappeared in time and the patient, up to 1896, felt well. In the year 1878 (five months after the injury) Professor Aretie, having discovered the bullet by means of an electrical apparatus, had already made an attempt to remove it, but did not succeed. Beginning in 1896, the patient commenced to feel tingling in the thumb and index finger, and then the two upper extremities and the lower extremities grew weak. He could not move his head in complete extension backwards. There appeared in 1898 a muscular atrophy of the four extremities, worse on the right side (difference of about one cm. in the circumference), spasmodic gait, paresis of lower extremities, reflexes of upper and lower extremities were exaggerated, spinal tremor, but sensation everywhere normal. Micturition and defæcation normal. Neither trophic nor vasomotor disturbances. The bullet was felt and extracted after incision of the soft parts.

What is remarkable in this case is the late meningo-myelitis phenomena (spasmodic paraplegia, atrophies, etc.) which appeared eighteen years after the traumatism, the patient remaining in good health during all of this long interval.

Case XXXIII.—Maughinas. *Ibidem*, p. 691. G. F., soldier, twenty-eight years old, injured May 2, 1897, by a firearm of small calibre. Point of entrance of the bullet was over the fourth zygomatic bone, point of exit behind the right ear, five centimetres below it. After the injury there appeared feebleness and numbness in the upper extremities, and a positive feebleness of the right lower extremity. There were inability to open the mouth and to take nourishment, contracture of the jaws, rigidity and difficulty in the movements of the nape of the neck. At the right of the neck, opposite the third cervical vertebra, was a painful prominence. The patient could not lie down, and protected the right head. Improvement was gradual. On June 18, movements of the fingers and opening of the mouth were better. Pain in the vertebral column persisted, at times very strongly. On July 1 he left the hospital with a weakness in the right upper and lower extremities, and a positive stiffness in the muscles of the nape of the neck.

Case XXXIV.—Kokoris. *Ibidem*, p. 692. B. C., aged twenty-two years, alcoholic, was injured April, 1897, in war, by a bullet which entered the body on a level with the sixth cervical vertebra, a little to the right of the median line, and passed out on the opposite side. The wound closed rapidly, and the patient felt nothing much until the beginning of the following year (1898), when his upper extremities began to get weak, and he began to experience, at the same time, pains in the nape of the neck. In March, 1898, M. Kokoris saw the patient, who could not use his upper

extremities for performing his work as a laborer (to dig, etc.). He found the scars of entrance and exit of the bullet. There was evident paresis of the upper extremities, mainly the right. Sensibility to pain was diminished on the right half of the nape of the neck, in the shoulder and on the anterior aspect of the upper extremities as far as the fingers of the hand on the same side. On the left side the sensation was normal. The patient felt tingling around the cicatrices in the nape of the neck, in the right shoulder as far as the elbow, and on the anterior aspect of the forearm of the same side. Reflexes normal. There appeared no disability of any of the other organs.

Case XXXV.—Papatheodoron. *Ibidem.* K. G., soldier, wounded May 5, 1897, in war, by a portion of an exploded shell, which entered the body through the right scapula on a level with the spine, passed to the left, after having fractured in its course the arch of the third dorsal vertebra. Immediately after the injury paralysis of the lower extremities and of the trunk occurred, beginning at the ribs corresponding to the injury. Also paresis of the rectum and bladder. Diminution of sensation was present up to the level of the third thoracic vertebra. After several days of treatment (antiseptic dressing, immobilization) the morbid phenomena began to recede, so that, on May 16th, sensation had positively returned, and certain movements, especially of the right foot, were restored. On May 17th the patient urinated voluntarily. Towards the end of the month, he could turn over in bed and later raise himself alone. He could also extend and flex his extremities, the movement of extension being more difficult than that of flexion. The feet were in a position of varus equinus. Sensation had likewise been restored. In this condition the patient departed for his home.

Case XXXVI.—Dascalachi et Elephtheriadis. *Ibidem,* p. 720. Man, wounded by a revolver shot, as a result of which the bullet lodged in the body of the eleventh dorsal vertebra, as the radiograph made by Dr. Javanovich demonstrated. The spinal marrow was found entirely severed by the bullet, and the patient died several days afterwards.

Case XXXVII.—Dascalachi et Elephtheriadis. *Ibidem,* p. 720. Fracture of the thoracic vertebra, following a revolver shot, in a young man of fourteen years. The result was fatal.

Case XXXVIII.—Chipault. *Ibidem,* II., 1903, p. 780. The correspondent of the *British Medical Journal* in South Africa observed a soldier who had received a bullet which entered underneath the tenth rib, on the left axillary line, passed in front of the vertebral column, probably grazing it, and came out on the right axillary line underneath the seventh rib. The shot had been fired at 600 yards and had made a perfectly clean wound, both at the point of entrance and at the point of exit. At the time of the injury, the effect most particularly noticed was the loss of motion of the legs. This gradually returned at the same time when there appeared in the lower extremities pains which became more and more intense, and which persisted.

Case XXXIX.—Gianelli. Chipault. *Ibidem,* iii, 1903, page 907. A man of twenty-one years, on July 4, 1897, received a revolver shot in

the left side. He immediately fell to the ground. In the night following, there was incontinence of urine. On the third day he had œdema of the lower extremities, sloughs, complete abolition of sensation and motion in the lower third of the thorax, the abdominal walls, and the lower extremities. The patellar, pedal, and cremasteric reflexes were completely abolished. Retention of urine with incontinence from overflow. On the 20th, incontinence of fæcal matter; on the 30th, a sacral bed sore. Death on the 12th of August.

At the autopsy, in addition to the cystitis, pyelo-nephritis and fatty degeneration of the organs were found. A comminuted fracture of the body of the third dorsal vertebra, where the bullet was imbedded, had compressed and softened the cord.

Case XL.—Grey. Gunshot wound of the spine. *Ibidem*, page 807. An Indian of twenty-two years, was shot by a rifle; when the ball struck him he fell paralyzed. It had penetrated the body three centimetres to the left of the eighth dorsal spinous process. The wounded man entered a hospital two and a half months later. He had œdema of the lower extremities (sloughs), complete paralysis of sensation and motion ascending as far as the seventh dorsal process and three fingers' breadth above the xiphoid appendix, abolition of reflexes, incontinence of urine and fæces. In the back was an oblique cicatrix, three centimetres long, ending at three centimetres outside of the eighth dorsal spinous process. An incision was made from the fourth to the eleventh dorsal process with two transverse incisions at the extremities, the arches of the seventh and eighth dorsal vertebra were removed, the bullet was found beneath and attached to the seventh process, its point facing directly into the spinal cord which was missing, scarcely a thread of connective tissue being present. Union was by first intention. In consequence of the operation the sloughs and the œdema disappeared, and the general condition improved, but two months later the patient began to deteriorate anew, and he was able to survive but a short time.

Case XLI.—Chipault. *Ibidem*, page 828. My most remarkable case is that of a young man who had received a shot from a revolver in the cervical region. The missile entered the left carotid region, came to stop in the skin of the axilla of the right side, after having in its course reduced almost to splinters the third and fourth cervical vertebræ, and leaving the cord exposed. Operation was quite difficult, because the missile having deviated from its course, had passed through the regions of the large vessels of the neck and the arm, scattering in its course débris of bone, clothing, and hair. Success was perfect. The patient recovered, retaining all of the movements of the head and of the neck, as in the normal state. He paid no attention to a slight atrophy of the muscles of the right shoulder, which disappeared almost entirely with proper gymnastic exercises.

Case XLII.—Justo. *Ibidem*, page 887. Gunshot wound of the cervical cord. Patient had been wounded in February, a revolver ball having entered third or fourth centimetres from the median line at the top of the superior border of the thyreoid cartilage. His first stay at the hospital was from March 31st to April 21st. At this time the patient

complained only of indefinite pains and walked perfectly. After his departure, his condition gradually changed. There appeared a complete paralysis of the lower extremities and an almost complete paralysis of the right extremities. Respiration had become altogether diaphragmatic. Tactile sensibility was retained over the entire body, but sensibility to pain was abolished in the lower extremities and in the trunk. It was greatly diminished in the hands and in the forearms. In the arms and shoulders there was a slight hyperæsthesia, also in the neck, and its inferior limit was two centimetres underneath the handle of the sternum. Later this hyperæsthesia zone was, during the night, the seat of copious sweatings. Thermic sensation was retained in the analgesic parts and exaggerated in the hyperalgesic parts. Patellar reflex was exaggerated, especially on the right side. Pupils were normal. There was incontinence of urine and constipation. An operation was performed on May 16th, resection of the fifth and sixth cervical laminæ. During the night of the following day, death occurred without any other precursory signs than much tossing about.

At the autopsy, there was found an abundant retropharyngeal purulent collection in continuity with the body of the fractured third cervical vertebra. The pus extended up to the dura mater. The bullet was not found.

Case XLIII.—Estes, *International Journal of Surgery*, April, 1906, pp. 132, et seq. The third case of myellorectomy was for a gunshot wound of the ninth dorsal vertebra. The bullet lodged in the spinal canal and completely severed the cord. I exsected about three quarters of an inch of the disintegrated cord and brought the dura and ends together as before. This case was the only one operated after Dr. Stewart's and Dr. Harte's case was reported. The operation was not done until the eighth day after the injury, as the woman and her friends persistently refused surgical intervention until the last minute. Myelitis had already developed, and the patient died.

The three following cases have been furnished to me by Dr. Warren S. Bickham, to whom I am indebted for the privilege of publishing them in this series:

Case XLIV.—W. D., young negro man, age not given, admitted to ward 4, Charity Hospital, New Orleans, November 3, 1896, with penetrating bullet wound one inch to left of median line of neck on level with upper border of thyreoid cartilage, ball lying beneath the skin of his back on the right side, between spine and scapula, on a level of the seventh cervical vertebra. There were characteristic paralytic phenomena of corl involvement of that level.

Laminectomy of sixth, seventh, and eighth cervical vertebræ was done. The ball had pierced the body of the seventh vertebra, completely and cleanly severing the cord, the ends being about three quarter inches apart. An unsatisfactory attempt was made to suture cord and membranes (meningomyelorrhaphy). Wound was drained and sutured. No improvement followed, and death occurred on November 7, 1896.

Case XLV.—E. H., negro man, age not given, was admitted to ward 4, Charity Hospital, New Orleans, December 12, 1896, having been shot on December 9 from behind by a .38 calibre ball, and presenting wound immediately over the sixth dorsal vertebra, with loss of motion in and hyperæsthesia of lower extremities, and other phenomena of cord involvement. These symptoms continued until December 15th, when cerebrospinal fluid flowed so freely that it penetrated the mattress and collected on the floor. On December 18th, laminectomy of the sixth dorsal vertebra was done and the bullet removed from within the spinal canal, where it lay beneath the lamina which it had pierced, firmly embedded and pressing against the cord, the contour of which it had markedly altered and the meninges of which it had slightly lacerated, thus allowing escape of cerebrospinal fluid. Record does not state how wound was treated, but states that healing was by primary union. Patient improved slowly but steadily, and left the hospital well on March 4, 1897.

Case XLVI.—S. M., male negro, age thirty years, was admitted to ward 4, Charity Hospital, New Orleans, November 22, 1897, shot by a pistol in the axillary line over the seventh rib, with paralysis of lower extremities, bladder, and rectum. (Presence of yellow fever had deterred patient's coming to hospital earlier.) No evidence of wound of pleura or lung. On November 23rd, after attempting to follow up suppurating tract of bullet, five spinous processes and their laminæ were exposed, corresponding with the suggested range of the bullet and the clinical phenomena. Two sets of laminæ were removed, and the cord exposed and examined without result, by which time patient's condition had become so serious that further steps were discontinued. The patient was infused, and the wound closed with drainage. He rallied and seemed better for about thirty-six hours, and then suddenly became delirious and died.

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THE TREATMENT OF CHRONIC OBSTRUCTION IN THE LARYNX AND TRACHEA.*

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The causes of chronic obstruction in the larynx and upper trachea are generally classified as follows:

1. Cicatrices of traumatic, operative, or pathological origin. When following tracheotomy the deformities may be due to (*a*) contraction of the posterior soft part of the trachea following separation of the anterior segments by the cannula; (*b*) dislocation of the cricoid forward and downward, especially when the fistula is through or close to it; (*c*) overriding of the cut anterior ends of the tracheal cartilages in too long a fistula; (*d*) inversion of the cut anterior ends of the tracheal rings, especially if the fistula is to one side of the median line; (*e*) contraction of the trachea around the cannula due to sloughing of the cartilages or rings; (*f*) spur formation posteriorly (as after enterostomy), especially in combination with contraction of the posterior soft portion of the trachea, or dislocation of the lower segment forward and the upper backward, or both.

2. Granulations around a cannula or intubation tube which are much less common than is generally believed. They have been described as occurring at the upper anterior angle of the fistula for a tracheal cannula, or opposite the usual fenestrum on the posterior tracheal wall, or at the lower end of the cannula or around its whole length, or about the upper or lower end of an intubation tube.

3. Hypertrophic subglottic laryngitis most commonly encountered in retained tube cases, less often in those with a retained tracheal cannula and almost always after diphtheria.

4. Prolonged and repeated diphtheritic inflammation, which must be exceedingly rare in these days of antitoxin, although it is

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worth noting that there is at least one case on record in which the exudate was demonstrable on the one hundred and fifty-first day.

5. Postdiphtheritic paralysis of the laryngeal abductors, which is sometimes described and must be very rare, but clinically is indistinguishable from the following:

6. Atrophy of the laryngeal abductors from disuse in retained tube and tracheotomy cases.

7. Obturation or compression of the respiratory tract by neoplasms is common and involves first the removal of the tumor and next the treatment of any concomitant or resulting cicatricial stenosis.

Of these "chronic hypertrophic subglottic laryngitis" needs some discussion. It was described by Köhl* as one of the common causes for retention of the tracheal cannula after acute diphtheritic stenosis long before the days of intubation, but at present is generally regarded with suspicion or mistaken for "granulations" or a cicatrix. I had the correct diagnosis first forced upon me by undertaking laryngotomies to remove these supposed granulations or cicatrices in cases of retained tubes. The gross appearance was that of a general velvety thickening of all the soft parts within the thyroid and cricoid cartilages, which bled easily, though there was no ulceration. The vocal cords and ventricles showed only as a slight tumefaction or dimple, and when the divided throid alæ were brought in apposition the hypertrophied mucous membrane completely occluded the laryngeal lumen. The futility of operation in such a condition is obvious and to elucidate its pathology, as clinically at least, it is one of the commonest causes of retained intubation tubes, I must repeat in full the findings I have been able to collect.

I am indebted to Dr. F. Tilden Brown for the first specimen, that of a child intubated at the Willard Parker Hospital June 28, 1896. Autoextubation, followed by closure of the larynx within a few minutes, necessitated tracheotomy about a month later. A year subsequently, or on July 31, 1897, as the stenosis was unchanged, Dr. Brown inspected the larynx through a thyrotomy at the Presbyterian Hospital. There was an even thickening of the soft parts throughout the whole interior of the laryngeal cartilages. There was no sodden or watery appearance such as might be produced by œdema, but the thickened tissue seemed firm and

* Archiv f. klin. Chir., 1887, Band XXXV., pp. 75 und 423.

vascular. Bleeding was easily excited by sponging. There was no sign of ulceration nor cicatrization, and the soft parts were apparently covered everywhere with epithelium. A vertical strip of tissue was then cut from the whole thickness of the soft parts on each side of the inner surface of the larynx. The pathologist of the hospital reported that these strips showed "infiltration with many small round cells, in places apparently granulation tissue. Over some thickened areas there is thin mucous membrane." It would be difficult to cut sections of such strips in a plane which would demonstrate the presence and integrity of the epithelium

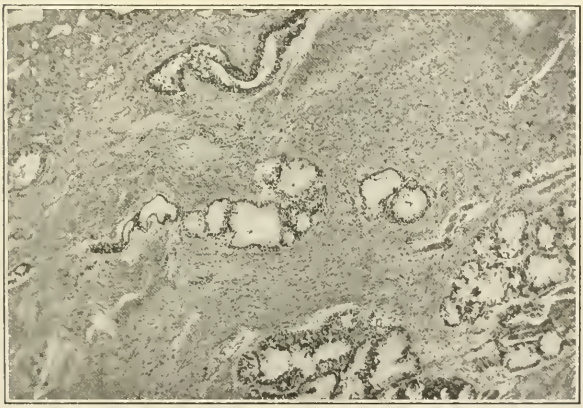

FIG. 1

Section through the middle of the larynx of Case VI., showing new connective tissue beneath the mucous membrane.

throughout, in other words, to prove by the microscope the absence of ulceration. But this may be inferred from the gross findings in spite of the use of the words "granulation tissue," which are intended to indicate a state of exudative and productive inflammation. This child died later of pneumonia.

The operation did not succeed in making respiration possible without the tracheal cannula.

I am indebted for the second case to Dr. Dillon Brown. This child was intubated by him for laryngeal diphtheria in the first week of February, 1897. The disease subsided, but the stenosis

persisted and necessitated "retention" of the tube. Respiration without it was barely possible for about an hour, but no longer, and finally after autoextubation at the end of a month from the time of first insertion asphyxia occurred. At autopsy the intralaryngeal soft parts were found to be evenly thickened to such an extent that the lateral portions of the larynx were in contact in the median line. The tissues were moderately soft and compressible enough to permit the introduction without undue force of the tube ordinarily employed for a child of this age and size.

Dr. W. N. Berkeley, the pathologist then in Dr. Brown's laboratory, cut some sections for me transversely through the

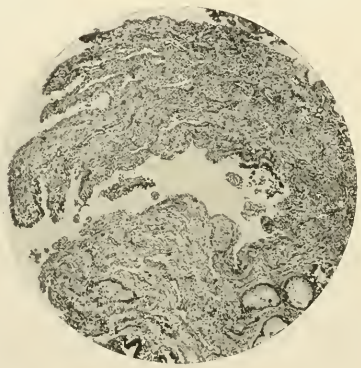

Fig. 2.

From Case VI., showing the polypoid outgrowths in the chronic hypertrophic laryngitis.

centre of the larynx and made the following report: "The mucous membrane, submucosa and remaining soft parts within the larynx appear much swollen and thickened by a growth of new cells. The vessels are numerous and congested. There is an irregular increase in the superficial epithelium and an hypertrophy of the subepithelial papillæ. There is no evidence of ulceration with loss of substance. The striated muscle fibres, both in front and behind the arytenoid cartilages, are present in the usual amount, but like all the other soft tissues, are infiltrated with columns of small round cells. In all probability this infiltration would interfere with or abolish the function of the muscles. The cartilage is normal."

The third specimen is from Case VI. and is one approaching a cure. This child had worn an intubation tube for nearly three years and had never been tracheotomized. Only during the latter two months of tubage (and her life) was natural respiration possible for even a few hours.

The gross appearance was one of smooth, velvety thickening of all the soft parts within the laryngeal cartilages. This thickening reduced the lumen of the larynx to about one-third of its natural size. Within the anterior portion of the cricoid there was an irregularly circular area about one-quarter of an inch in

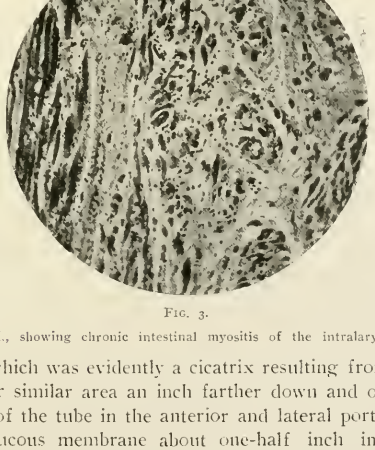

FIG. 3.

From Case VI., showing chronic intestinal myositis of the intralaryngeal muscles.

diameter, which was evidently a cicatrix resulting from an ulcer, and another similar area an inch farther down and opposite the lower end of the tube in the anterior and lateral portions of the tracheal mucous membrane about one-half inch in diameter. These areas did not constrict the lumen of the larynx or trachea, but, on the contrary, seemed slightly depressed below the surrounding soft tissue. Ulceration had therefore occurred at these points and the healing had increased rather than decreased the lumen of the passage.

To make clear the explanation of the treatment proposed I must anticipate by stating that by a "normal" tube is meant one of the ordinary O'Dwyer instruments as originally made by George Ermold & Co. in seven sizes, usually distinguished as suitable respectively for a child one year old or younger—two, three, four

to five, six to seven, eight to nine, and ten to twelve years of age. For adults the sizes run from one to ten inclusive. By "special" tube is meant one of the same length as the normal, but in which the neck or constricted portion just below the head or the part below the retaining swell is greater in the transverse diameter generally by 1/32 inch or some multiple of this amount. The "retaining swell" or bulging central portion of the tube must at the same time have a transverse diameter at least 3/32 inch greater than the neck or the tube will certainly be coughed out. In cases of hypertrophic laryngitis which are totally unable to breathe without a tube this difference is too slight and a differ-

FIG. 4.

Special tubes in Case VI. A, normal tube.

ence of 4/32 or even 5/32 inch may be required. Life will often depend on this 1/32 inch.

By a "plugged" tube is meant one fitted with a plug made to screw accurately into a perforation in the anterior wall of a normal or special tube through a tracheal fistula. The plug is perforated at its inner end by a hole which exactly corresponds in size with the lumen of the tube. The plug is provided with a retaining pin, which, when the plug is in place, prevents it from rotating and so obstructing in the slighest way the lumen of the tube. By previous measurement the tube has a hole bored in its anterior wall, which when the tube is inserted lies opposite the tracheal fistula. Into this hole the plug is then screwed and "pinned" at right angles to the tube. A cap is then screwed down

over the outer end of the plug to hold the pin in place. By a "clamped" tube is meant one fitted with a detachable pair of metal clamps made on the principle of an obstetric forceps. In the sides of the tube are cut grooves, which, when the instrument is in place, lie at the level of the tracheal fistula. Through this the clamps are made to grasp the tube and are then held in place by a collar screwed down around them until the collar touches the tube. The plugged or clamped tube is a guarantee against autoextubation and at the same time keeps the tracheal fistula from closing.

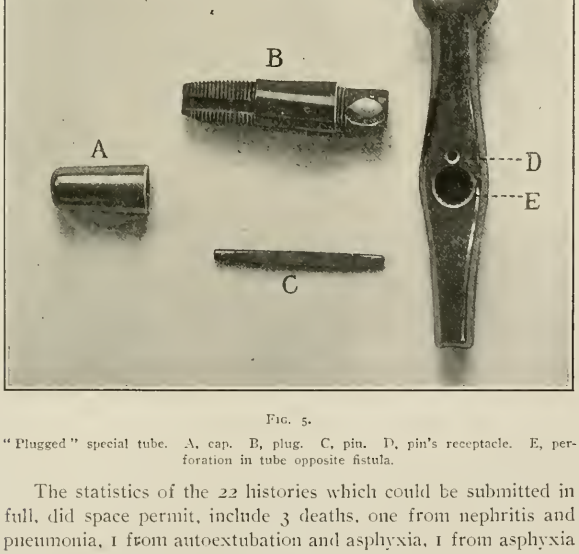

Fig. 5.

"Plugged" special tube. A, cap. B, plug. C, pin. D, pin's receptacle. E, perforation in tube opposite fistula.

The statistics of the 22 histories which could be submitted in full, did space permit, include 3 deaths, one from nephritis and pneumonia, 1 from autoextubation and asphyxia, 1 from asphyxia due to the clogging of a metal tube; 2 failures to cure; 7 still under observation. I do not consider a case cured until at least a year has elapsed after the last intubation without the appearance of any signs of stenosis. Of these 7 cases 3 are able to breathe naturally for a few hours or days, although the obstruction when

first seen was complete; 2 (adults) have been from one to three weeks without a tube, which was only reinserted when the laryngoscope showed evidence of beginning recontraction (these 5 are therefore still wearing tubes); 2 apparently cured, but the requisite year has not yet passed; 10 cured, that is, they have remained free from any obstruction for at least one year and some for four or five years. All of these have voices which range from perfect to hoarse.

Case I.—*Stenosis due to hypertrophic laryngitis of unknown origin; thyrotomy and some cicatricial contraction added to the pre-existing difficulty; cured permanently at the end of four weeks by intubation with the largest normal tube which could be passed through the larynx with slight force.*

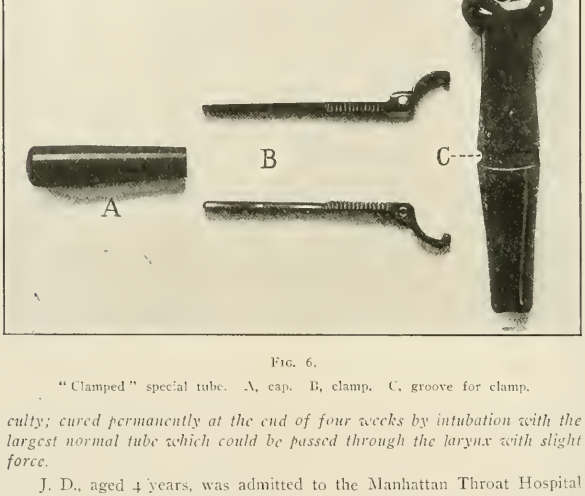

Fig. 6.

"Clamped" special tube. A, cap. B, clamp. C, groove for clamp.

J. D., aged 4 years, was admitted to the Manhattan Throat Hospital under the care of Dr. Hope in March, 1902. He presented the general characteristics of rickets and a history was given of frequent attacks of spasmodic dyspnœa since birth. These had lately increased in frequency and severity and for the past week or two the difficulty in breathing was considerable and constant. Laryngeal examination was impossible. Tonsils and adenoids were removed without effect on the laryngeal condition, and therefore on April 16th a low tracheotomy was carefully performed in the median line with the object of giving rest to the larynx. During the operation the thymus gland was exposed and noted to be very

large, but not in any way compressing the trachea or obstructing respiration. June 10th, no improvement followed and a thyrotomy was undertaken. This revealed a general thickening of all the soft parts within the larynx. There were no ulcerations, nor cicatrices, nor tumors. As the condition appeared to be a simple hypertrophic laryngitis, nothing

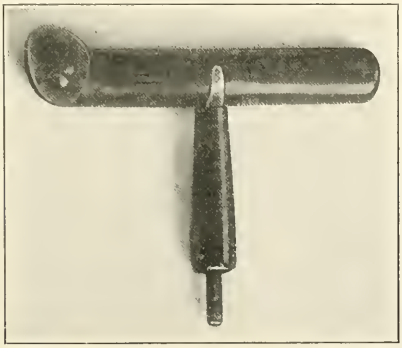

Fig. 7.

Clamped tube.

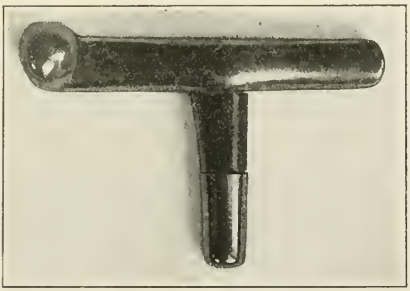

Fig. 8.

Plugged tube.

further was attempted, and the wound closed with the tracheal cannula in the lower angle. (The above history was given to me by Dr. Hope.) Some improvement followed, but on August 16th he was transferred to Gouverneur Hospital, where, under chloroform, I found that respira-

tion without the cannula, though barely possible for one or two gasps, was not compatible with life. Cicatricial contraction was present, but needed only slight dilatation and the largest rubber tube which could be inserted without force was placed in the larynx. September 9th, the intubation tube was removed on this date, and as no dyspnœa appeared, it was not reinserted. July 18, 1904, the child was inspected on this date and found entirely well, with a normal voice. There had been no recurrence of any dyspnœa.

Case II.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; cured by prolonged intubation with the largest normal rubber tube which could be pushed through the larynx with slight force; duration of proper treatment nine months; some adductor spasm.*

M. W., aged 4 years, was admitted with diphtheria to the Willard Parker Hospital and intubated October, 1896. A month later autoextubation began to give trouble, but by using the largest tubes which could be introduced without force this subsided. The tubes were frequently changed, and as the stenosis persisted and was complete, attempts were made to overcome it by decreasing the size of the tubes, even down to the No. 2 normal. In May, 1897, he was transferred to the Manhattan Eye and Ear Infirmary for the stenosis, but was not benefited. He then contracted scarlet fever and survived it and came to the Gouverneur Hospital in October, 1897, where again tubes as small as would stay in the larynx were tried, with the idea of minimizing all traumatism. As a natural result one of these slipped into the trachea and was only extracted through the larynx with the greatest difficulty. In March, 1898, as the stenosis was still complete, an attempt was made to produce a pressure atrophy by inserting a tube as large as the larynx would bear and an eight-to-nine tube was inserted with some slight violence. This was worn, with the usual monthly changes for cleansing, until November, 1898, when on removal the dyspnœa was found to have entirely disappeared. During the following first few days there were frequent attacks of coughing followed by inspiratory dyspnœa, and though in each asphyxia seemed imminent, the administration of morphine checked the spasms and the difficulty gradually passed off. A year and a half later when the child was last seen the voice was somewhat husky, and after coughing or violent exercise there was apt to be stridulous inspiration, but there was no dyspnœa.

Case III.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; failure with normal and "special" rubber tubes; success with a "special" metal tube; cured after eleven months of proper treatment.*

K. T., aged 2 years, was intubated on account of diphtheria in September, 1900, at her home. The stenosis remained complete, in spite of frequent changes of the tube, and she was first seen by me in January, 1901. Absolutely no air passed through the larynx when the tube was removed and considerable expedition and care was necessary to prevent asphyxia. There was no bleeding and no necessity of using force in intubating with the normal tube of proper size, and therefore, clinically, there was no ulceration and no cicatrix. The parents refused to leave this

child in the hospital, and I only saw her when the breath became foul, or about once a month when the tube was changed. At first the plan of treatment was expectant, using only the proper-sized normal tubes. In June, 1901, a very little air was found to pass through the larynx, but later even this failed. In September I began using normal rubber tubes as large as the larynx would admit without undue force. There was no change, and in January, 1902, a "special" rubber tube, with the diameter of the neck larger than the normal, was tried also without success. In March a special tube of bronze, heavily gold plated, was substituted for the special rubber tubes. The neck and retaining swell of this tube were three thirty-seconds of an inch larger in the transverse diameter than these parts of the normal tubes. With this metal tube some improvement was noted each month, and on August 1, 1902, the child was dismissed cured. In July, 1904, the child was entirely well and had had no recurrence of dyspnœa, and the proud mother informed me she had recently taken a prize for singing in the public schools. There was nothing abnormal apparent in the voice. The danger of sudden death, which this child survived during the long period of twenty-three months in which she wore a tube, is obvious.

Case IV.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; death from autoextubation.*

A. V., aged 10 years, was intubated for diphtheria during October, 1900, at the Willard Parker Hospital. The tube was "retained," and in June, 1901, she was transferred to Gouverneur Hospital. Under chloroform it was found that after removal of the tube absolutely no air passed through the larynx. It closed tight instantly. The operation produced no bleeding, there were no granulations perceptible, and no force was required to insert the eight-to-nine tube, which was suitable for the size and development of the child. The condition was, therefore, clinically that of a chronic hypertrophic laryngitis. A large "special" tube of the eight-to-nine length was therefore inserted, and as it was worn without any sign of difficulty or coughing for five weeks, the child was allowed to go out and home one afternoon for her birthday. She was given some unusual article of diet and vomited and was asphyxiated during a tracheotomy hastily performed by a doctor summoned from the neighborhood. No autopsy was permitted.

Case V.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; death from asphyxia due to clogging of a metal tube.*

R. R., aged 3 years, was intubated for diphtheria at the Willard Parker Hospital in December, 1900. The tube was retained, and in August, 1901, she was transferred to the Gouverneur Hospital. In changing the tube the same conditions were found as in Cases A. V. and K. T., that is, the larynx closed immediately and completely when the tube was withdrawn. This is a typical picture of the hypertrophic laryngitis which follows a diphtheria requiring intubation. The diphtheria subsides, but the stenosis persists indefinitely. As large a metal tube as could be passed without undue force was inserted at this date. This was changed about every four or five weeks, the same tube being re-

placed each time, although the constant use caused some roughening of the exterior and erosion of the gold plating with a kind of calcareous deposit. This stony material was scraped off the tube each time, but it gradually became less smooth than it should be. In November, 1901, it was changed on account of noisy respiration one week after the last previous insertion, a much shorter period than had usually been necessary for the tube to become foul, but as improvement in the larynx was quite marked and there happened to be no other similar metal tube at hand, the roughened one was replaced with the remark that a new one would be obtained in a day or two. That night the tube became clogged, and between attempts at removal and tracheotomy by the house surgeon the child was asphyxiated. No autopsy was permitted. A cure in this case seemed close at hand, as at the last change respiration, though somewhat difficult, was possible for some time.

Case VI.—*Stenosis due to hypertrophic laryngitis following intubation persisting unchanged for two and a half years; some improvement during the next six months with "special" rubber tubes; adductor spasm evident; more marked improvement with special metal tubes; death ultimately from nephritis and pneumonia.*

G. S., aged 3 years, was intubated for diphtheria at the Willard Parker Hospital in July, 1902. The stenosis persisted and the tube was worn continuously thereafter with frequent changes until February, 1904. On this date a special rubber tube, of the four to five length, was inserted, having the neck and retaining swell one-thirty-second of an inch larger in diameter than the normal measurement. During the change absolutely no air passed through the larynx, no bleeding was produced, and no force was required for intubation. The condition was therefore clinically one of chronic hypertrophic laryngitis. February 20th the tube was coughed out for the first time in her history and also for the first time in two years she breathed three days unassisted. Then the tube had to be reinserted on account of increasing dyspnœa. This comparatively slow recurrence of the stenosis often occurs during recovery. It is too rapid to prove an error in diagnosis or that the condition is cicatricial. February 28th a larger special rubber tube was inserted, and special tubes of increasing size were used with infrequent changing until July 8th. At this change, when the largest of the special tubes had been worn a month, the condition seemed as bad as ever, and acting under the belief that a spasm of the adductors of the larynx might have something to do with the difficulty a normal four-to-five tube was inserted until July 16th. On removing the last or normal tube I found the breathing, though somewhat difficult, was fairly good and hence the supposition that the obstruction at the last change was due to a cramp of the adductors and atrophy of the abductors seemed correct. I therefore returned to the special metal tube, which had produced the first improvement in March. July 27th this tube was removed and cleaned by the house surgeon, as it seemed to be foul and producing a cough. Respiration, though difficult, was possible. In August a nephritis developed (and this was followed by a chronic pneumonia, which at autopsy was pronounced by the pathologist to be simple and not tuberculous). August 26th the tube was removed and

natural respiration for the first time was easily possible, but the dyspnœa gradually returned and on August 29th the tube had to be replaced. The child continued to gradually lose ground and finally died from the pneumonia on September 12th. The tube was not removed until after death, but then the larynx appeared open enough for respiration to have been carried on unassisted. The report on the specimen will be found in the first part of this paper.

Case VII.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; complicated by cicatrices resulting from several laryngotomies and tracheotomies; obstruction from spasm of the adductors; also from granulations; cured by two and a half years of treatment with "special" tubage; duration of stenosis, six years.*

M. M. was admitted to the Willard Parker Hospital June 2, 1896, at the age of 3 years. She had been intubated six days previously for diphtheria. The stenosis persisted and on account of frequent and dangerous autoextubation she was tracheotomized July 13th. The stenosis still persisted and intubation was again tried. Astringents like alum applied on the outer surface of the tube to the laryngeal mucous membrance, as advised by Dr. O'Dwyer and afterward curetting the larynx through the mouth, were tried without success. In October, 1896, transferred to the Metropolitan Throat Hospital and thyrotomy performed by Dr. Wagner. Some thickened tissue removed from the interior of the larynx and the wound packed. Breathing through the tracheal cannula. The only pathological change noted during the operation appeared to be a general thickening and hypertrophy of all the intralaryngeal soft parts. In October, 1898, another thyrotomy, performed by Dr. Hope, with division and excision of numerous cicatrices. In January, 1900, the patient, then seven years old, was transferred to Gouverneur Hospital still breathing through a tracheal cannula. Under chloroform a much deformed larynx, which barely admitted one of the smallest urethral sounds, was dilated from below through the tracheal fistula with urethral sounds up to No. 25 F. There was a "spur" opposite the fistula and the entrance to the trachea below was contracted around the cannula; this was also dilated like the larynx. A ten-to-twelve normal intubation tube was then inserted and subsequently changed every four or five weeks for cleansing. In May, 1900, the tracheal fistula finally closed and so cut off all respiration except through the larynx and the stenosis was still marked. At this change I made several attempts at intubation and failed on account of the deformity and displacement of the larynx due to the numerous cicatrices. The consequent traumatism produced complete obstruction and asphyxia was barely avoided by a hasty tracheotomy, after which intubation was finally accomplished. In July, 1900, as a preliminary to changing the tube and to avoid the previous danger, I again punctured the trachea and then intubated without difficulty. This largest-sized tube was changed at intervals of four weeks without perceptible gain and therefore with the object of obtaining the utmost dilatation a "special" tube was employed of the same length as the normal but of much larger diameter. This was worn with the usual changes until January 15, 1901, when it was removed and a cure seemed at last obtained, but five days

later at night there was a sudden attack of dyspnœa and the child was barely saved from asphyxia by another hasty tracheotomy by the house surgeon. Some infection followed with recontraction of the cicatrices, and not until March was intubation again practised. A little later dyspnœa began to reappear with the tube in place. An exploration revealed exuberant granulations at the base of the epiglottis overtopping the tube and blocking its lumen. In July another thyrotomy had to be performed to gain access to the obstruction and the granulations were then scraped and cut away. A normal tube with a small head to avoid pressure on the ulcerated surfaces was inserted at the close of the operation. This was worn until no more bleeding occurred at the periods for cleansing. In November the special tube with the large diameter was again used and worn with occasional removals for cleansing until February, 1902, when a cure seemed obtained, but that night there was a sudden attack of dyspnœa and another tracheotomy by the house surgeon. On the following day the large calibred special tube was reinserted and worn until May. Then a normal tube of the eight-to-nine size was substituted for two weeks, and in June the patient was at last dismissed cured. She has remained well and free from dyspnœa ever since and even has a good though husky voice. She is at present attending school (October, 1905).

Case VIII.—*Stenosis due to hypertrophic laryngitis following intubation for diphtheria; complicated by the cicatrices of several tracheotomies and by adductor spasm; cured permanently after treatment during eleven months.*

C. S., admitted at the age of 5 years, with diphtheria, May 20, 1899, to the Willard Parker Hospital and intubated. The tube was retained and the stenosis remained complete, with the exception of the days from June 15 to 23, 1899, when the child was able to breathe unassisted, though with difficulty. In December, 1899, the patient was admitted to Gouverneur Hospital wearing a four-to-five normal tube, without which respiration was absolutely impossible. On January 25, 1900, under chloroform to obtain quiet, the largest normal metal tube (an eight-to-nine) which could be passed through the larynx without undue force was inserted. This was changed at intervals of four or five weeks for cleansing until March 29th, when a cure seemed obtained. Respiration continued normal for about a week; then on April 5th there was a sudden attack of dyspnœa at night, and the house surgeon being unable to intubate, performed a hasty tracheotomy, which saved the child's life. The tracheal cannula was removed on April 15th. No dyspnœa appeared and the fistula was allowed to heal. There had, however, been some infection and an abscess of the lymph nodes was opened in each supraclavicular fossa. In May, on account of gradually increasing dyspnœa, intubation was attempted, but the tubes were arrested at the site of the tracheal cicatrix. The manipulations and consequent traumatism nearly produced asphyxia and a hasty tracheotomy below the cicatrix was necessary. The cicatrix was then dilated from below up with urethral sounds and a normal eight-to-nine rubber tube inserted. This was changed at intervals of about five weeks until October 1, 1900, when a cure seemed obtained. A couple of

weeks later the respiration began to become noisy, and on October 13th an eight-to-nine tube was reinserted and finally removed on November 20, 1900. Since then there has been no recurrence of the dyspnœa and a fairly good voice is now present.

Case IX.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; thyrotomy followed by sloughing of the cartilages; permanent cure after three months of proper treatment.*

I. B., aged 4 years, admitted with diphtheria to Willard Parker Hospital and intubated in December, 1896. The stenosis persisted and necessitated retention of the tube. Frequent and dangerous autoexubation began, and to prevent asphyxia tracheotomy had to be performed on February 20th. A month later intubation with a normal four-to-five tube was practised with the tracheal cannula in place. No autoextubation occurred, so the cannula was removed and the fistula allowed to heal. The complete stenosis persisted and she was transferred to Gouverneur Hospital October 15, 1897. A week later the tube was coughed out and a hasty tracheotomy performed by the house surgeon. In November the cannula was removed and intubation again continued. January 10, 1898, as respiration was impossible without the tube, I explored the larynx by a median vertical incision through the thyroid and cricoid cartilages. The tissues were thickened and soft throughout the whole interior of the larynx, in which there were no ulcerations and no cicatrices. By bringing the parts together it was found that the hypertrophied mucous membrane completely occluded the lumen of the larynx. With the idea of producing a vertical cicatrix on each side of the larynx and so retracting the swollen parts I passed a silk suture through the cartilage and thickened mucous membrane on each side and tied it on the outer surface of the corresponding thyroid ala. This retracted the tissues sufficiently to allow easy breathing and the larynx was closed, but infection followed and in the course of the next few weeks apparently the greater part of the thyroid and cricoid cartileges sloughed out. April 20th the patient was discharged seemingly in a hopeless condition and condemned to the permanent use of a tracheal cannula. In August the patient returned with a dense cicatrice occuping the position of the larynx, but under chloroform with some difficulty a probe was passed from the tracheal fistula up into the mouth. This was followed with sounds of increasing calibre until the larynx and the entrance to the trachea had been dilated up to No. 24 F. A normal four-to-five tube was then inserted, the cannula removed and the fistula allowed to close. On October 1st the tube was removed and a six-to-seven normal tube substituted. The stenosis was still nearly complete. At the next change, in November, natural breathing was found possible and the tube was left out. For the first two weeks there were several attacks of inspiratory dyspnœa, especially at night, but under the administration of a little morphine these gradually disappeared. Since then the cure has remained permanent and the voice is fairly good.

Case X.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; complicated by cicatrices due to numerous tracheotomies; pneumonia as a complication of intubation; apparent cure*

followed by reconstruction of the tracheal cicatrices. This and Cases XI. and XIII. illustrate some of the worst and most discouraging features of extensive cicatricial deformity; apparently cured, but still under observation.

M. R., aged 3 years, was admitted with diphtheria to the Willard Parker Hospital on February 2, 1899, wearing an intubation tube which had been inserted two days previously. Respiration without the tube was impossible for more than a few moments. The larynx seemed to close immediately after removal of the tube, but no difficulty was encountered in its insertion and no bleeding occurred, and therefore, clinically at least, the condition was one of hypertrophic laryngitis. The frequent and dangerous autoextubation, which began soon after admission, required tracheotomy on February 18th. As the stenosis persisted intubation was again attempted at intervals of a month, but no tube could be retained in the larynx for more than a short time. The child was sent home in June breathing only through the tracheal cannula. The patient was examined under chloroform at the Gouverneur Hospital in October, 1899, and the stenosis found to be complete. The fistula was just below the cricoid cartilage. The stricture was densest just above the fistula; the intralaryngeal soft parts seemed to offer no resistance to the dilatation which was begun with a uterine sound entered through the fistula and followed by urethral sounds of increasing calibre until No. 21 F. was reached. So much force was required for the passage of this size that there seemed danger of tearing into the œsophagus, and a normal four-to-five intubation tube, though small for this child, was inserted with considerable violence. No reaction, however, followed this rather rough treatment, and ten days later under chloroform a six-to-seven normal metal tube was inserted without difficulty. This was changed and replaced after cleansing at intervals of four to six weeks with steady improvement. During November it is worthy of note that the child went through a rather sharp attack of pneumonia, the course of which did not seem to be unfavorably affected by the intubation tube.

On December 4, 1899, as a cure seemed obtained, the tube was left out permanently and the child dismissed.

The child was readmitted to the hospital August 1, 1902, suffering from severe dyspnœa, which was first noticed some two weeks previously. An attempt at intubation demonstrated that the larynx was free and the stricture opposite the tracheal cicatrix. The manipulation speedily produced complete obstruction and asphyxia was barely averted by a hasty tracheotomy through the lower end of the old scar. The dense and tight stricture was then dilated through this wound with urethral sounds until a normal eight-to-nine intubation tube could be easily inserted. This was worn with the usual monthly changes, the fistula being allowed to heal and with steady improvement until December, 1902. Then, as a cure seemed obtained, the child was again dismissed without the tube.

The patient was readmitted August 20, 1903, with a history of gradual recurrence of the dyspnœa, and as it did not seem severe, I attempted to intubate, and, as in the previous year, nearly killed the child, who was only saved at the last moment by another tracheotomy. After the usual dilatation with urethral sounds a special tube of the eight-to-nine length,

but with a retaining swell as large as the normal calibre of the trachea, was inserted. The fistula was allowed to heal, but the cicatrix resulting was some two inches long, and fearing recontraction the tube was allowed to remain (with monthly changes) until December 1, 1903. As respiration was then free the tube was left out with orders to report for inspection at regular intervals. But the parents failed to obey, and on September 1, 1904, the child was brought in showing considerable difficulty in respiration. Under chloroform, as the smallest tube which could reach the stricture in the trachea (six-to-seven) did not pass it, another tracheotomy was carefully performed in the centre of the old cicatrix. This was very dense and contracted the tracheal lumen, so as to barely admit a No. 12 F. sound. The stricture was then rapidly dilated up and down to No. 32 F., and the large special tube used before was inserted. On September 3 pneumonia developed, but subsided on the eighth day.

On November 6, 1904, the tube was coughed out, and as respiration was free it was not reinserted. Nevertheless, a recurrence was expected, and regular inspections at intervals of a week soon revealed slightly noisy respiration, and therefore, on November 26 a ten-to-twelve normal tube was inserted, and a week later a "special" tube, with the retaining swell and lower end having fully as large a diameter as the estimated proper calibre of this child's trachea. May 24, 1905. Since the last note the tube has been changed and cleansed once a month, and on this date the child appeared with his older sister, who said he had coughed up and *swallowed* his tube some three weeks previously. On examination I found the larynx open and with no sign of stenosis. Insertion of a tube similar to the one swallowed shows that no recontraction has occurred, and at this date (October 15, 1905) the child appears to be cured, although no positive statement to this effect in a cicatricial case is possible until at least a year has elapsed.

Case XI.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; complicated by cicatrices due to a tracheotomy for the relief of autoextubation; treatment with a clamped and then a plugged special tube and excision of the tracheal cicatrix; the use of the straight tracheal cannula to forestall adductor spasm after removal of the tube; still under treatment.*

K. M., aged 10 years, was admitted in October, 1903, to the Riverside Hospital for diphtheria and intubated. The diphtheria subsided, but the stenosis persisted. The tube was changed about once a week. The obstruction remained complete, and as frequent and dangerous autoextubation began in December, a tracheotomy was considered the safest method of treatment, and on December 13, 1903, was performed.

She was transferred to Gouverneur Hospital on May 29, 1904. There under chloroform it was found that the larynx was completely closed to inspiration, but apparently a very little air could pass through it in expiration. Sounds, beginning with No. 10 F., were passed up through the larynx and down into the trachea until No. 35 F. was easily admitted. There was a moderate "spur" opposite the fistula in the posterior tracheal wall, and around this spur and the fistula, especially just above it, there was a considerable cicatricial contraction. The most resistance was en-

countered just below the cricoid cartilage, but the intralaryngeal soft parts beyond seemed only thickened and soft. The fistula appeared to be through the second and third tracheal rings and in the median line. The child, though healthy and fat, was small for her age, and therefore a large special tube of only the eight-to-nine length was inserted. Within the next ten days this was coughed out several times, and the house surgeon, failing to replace it, had to dilate the fistula and insert the cannula. Without the tube no air could pass through the larynx. June 15, 1904, a large "clamped" rubber tube of the eight-to-nine length was inserted and worn without discomfort. This was changed about once in six weeks.

At the change on November 2, 1904, the breathing, though easy, showed some laryngeal "stridor," and therefore a still larger "plugged" metal tube was inserted. This had to be pushed quite vigorously to make the retaining swell pass the cricoid ring, but then, evidently, produced no undue pressure.

In the next week this tube had to be changed twice for cleansing, but at the second removal the interior of the tube was made smooth by dragging through it a piece of gauze well greased with vaseline. No further clogging occurred and the tube was finally removed on November 27, 1904.

Though respiration was perfectly free, a straight cannula, one-eighth inch in diameter, with its lumen occluded by a cork at the outer end, was inserted to keep the fistula open in case of spasm of the laryngeal adductors; this straight instrument does not displace the trachea as the ordinary curved cannula does and still maintains the potency of the fistula in case of accidents.

During the night of December 1, or about four days after removal of the intubation tube, there was a sudden attack of dyspnœa accompanied by spasmodic cough, and the patient was barely saved from asphyxia by the house surgeon, who found the cannula had slipped out of the fistula on to the neck. After reinserting the cannula there was immediate relief, and the following morning respiration without the cannula was perfectly natural. This was a typical "adductor spasm." A few days later the cannula was discarded and the fistula allowed to heal.

On December 8, 1904, slightly stridulous breathing was detected and an attempt at intubation met with some resistance in the upper aperture of the larynx. A little bleeding followed insertion of the tube, and though no laryngoscopic examination was tolerated, it was believed that an ulcer had been produced in the region of the vocal cords by the long-continued use of the large plugged tube removed in November, and that this ulcer in healing had caused the vocal cords to adhere throughout part of their free borders, or that a species of cicatricial diaphragm had developed in the neighborhood. There was no obstruction in the region of the tracheal cicatrix.

On December 20, 1904, this normal tube was removed, and as there was no bleeding and no sign of obstruction, a cure seemed obtained. About a week later, however, this child began to suffer from a rather severe typhoid fever, the infection apparently being due to the careless use of a thermometer by an inexperienced nurse. During the third week of the disease the breathing became a little noisy, and to forestall any aggravation of the symptoms I inserted the rather large special tube she had

worn in the early part of the winter. Though some resistance was felt in the neighborhood of the tracheal cicatrix, I believed the very slight dyspnœa was caused more by the typhoidal condition of the mouth than by a recurrence of the cicatricial stenosis. The child was at this time very ill. Nevertheless she recovered, and in the last week of January, when the temperature became normal, this large special tube was removed. But an hour later there was a sudden very severe attack of dyspnœa (adductor spasm), and I barely saved her from asphyxia by intubation with a normal ten-to-twelve tube.

On February 11, 1905, this tube was removed and respiration found normal, but a week later a test with the same tube demonstrated that the tracheal cicatrix had again contracted to such an extent as to admit at first only a six-to-seven tube. After dilating the ten-to-twelve tube was reinserted for ten days. Another test a week later, on March 1, showed that recontraction had again occurred, and as the stricture seemed to be in the nature of a thin diaphragm in the neighborhood of the cicatrix over the old tracheal fistula, I decided to explore and under chloroform opened the trachea again, carefully dissecting away all cicatricial tissue and found and exposed the cut anterior ends of the tracheal rings and brought them into accurate apposition over the normal ten-to-twelve intubation tube. There was quite a mass of fibrous tissue in the site of the old fistula which evidently interfered with the cartilaginous support of the anterior portion of the trachea and seemed to protrude into its lumen. Primary union was obtained, and on March 10, 1905, the tube was removed, but on the following day a recurrence of the dyspnœa again necessitated intubation, which will now be continued until a year has elapsed. The condition of hypertrophic laryngitis has entirely subsided and the child can breathe for some days without a tube, so there is no danger from autoextubation, and with long-continued dilatation there is every reasonable prospect in the course of months of obtaining a permanent cure. This child was examined with the laryngoscope on October 10, 1905, and practically no abnormalities beyond some congestion of the vocal cords could be detected. The voice, though hoarse, was good. She goes to school and is for all practical purposes well, but the tube is still worn and will be continued until next March, as this long-continued dilatation seems the surest way to provide against subsequent recontraction.

Case XII.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; complicated by cicatricial deformity the result of tracheotomy and subsequent division of at least part of the cricoid; adductor spasm evident; no improvement with the use of hard-rubber "special" tubes; marked improvement after wearing a metal "special" tube; still under treatment.*

E. McW., aged 6 years, had diphtheria and was intubated on October 28, 1903. Swelling around the head of the tube overlapping it caused dyspnœa and required a tracheotomy on November 5. Subsequently it was found that respiration, except through the cannula, was impossible.

Exploration under chloroform January 9, 1904, by prolonging the fistula up a short distance in the median line, revealed nothing but a general thickening. As the operation had to be abandoned on account of

threatened death from the anæsthetic, the findings were not very clear, but there was nothing to suggest ulceration or granulation.

On January 29, 1904, the patient was transferred to my care and under chloroform I enlarged the fistula, which seemed to be through the first one or two tracheal rings and at least the lower half of the cricoid, slightly downward and then found a considerable spur posteriorly opposite the fistula and around it the trachea contracted, so as to barely admit a No. 10 F. sound. Beyond this point there seemed no resistance, although after dilatation up to No. 30 F. no air could pass the larynx. There were no exuberant granulations. In other words, the condition was one of deformed and contracted trachea, with a hypertrophic laryngitis. An eight-to-nine normal tube was then inserted.

About once a month thereafter this was changed and a special rubber tube inserted of this length, but with the neck and retaining swell each time one thirty-second of an inch greater in diameter than that of the tube last used. At the change in June, as no improvement seemed to have occurred, ether was administered to determine whether an adductor spasm had anything to do with the obstruction, but even under the anæsthetic no air could pass the larynx, and some expedition was required in taking out and reinserting the tube. A sharp attack of pneumonia followed this experiment and for a time the child's life was in great danger, but recovery eventually took place.

At the change of tube on October 1, 1904, as no improvement had occurred, a special metal tube was inserted.

Removal of this metal tube on October 30th showed apparently the same persistent complete stenosis, and with the intention of putting in a "plugged" tube to escape the accident and probable fatality of possible autoextubation, chloroform was administered and when completely anæsthetized, the tube removed. Much to my surprise normal and easy respiration immediately followed. The metal tube seemed to have caused the hypertrophic laryngitis to subside and the obstruction following the removal of the tube without the anæsthetic was evidently due to spasm of the laryngeal adductors. But as I feared a possible later recurrence of the swelling the metal tube was replaced and, with the usual changes, is still continued.

In December, 1904, this child contracted whooping-cough, and it is worthy of note that no autoextubation took place and the spasms of coughing seemed to be borne more easily than they were by her two younger sisters, who were afflicted at the same time. In the latter part of January, 1905, she developed a rather bad case of measles, which subsided in about the usual time without complications.

In May, 1905, chloroform was again administered, and, after removal of the tube, respiration was found to be natural. The child was thus allowed to recover from the anæsthetic and then spoke in almost a natural voice for the first time since the diphtheria in October, 1903. The nausea and retching which followed the anæsthetic seemed to produce some dyspnœa, and I therefore replaced the tube. In spite of the evident absence of stricture very little air could pass the larynx during the succeeding monthly change of tube. Adductor spasm and fright are chiefly responsible, but there is still some of the hypertrophic condition

in the mucous membrance. It is, however, subsiding with the use of the large special metal tubes, as in September chloroform was again given, and, after recovering from the anæsthetic, the usual retching and coughing produced far less dyspnœa than ever before. Although I cannot yet report a cure, this case is interesting at least as showing the vicissitudes which one unfortunate child can survive. She is still wearing a tube, but attends school and is to all appearances healthy and strong.

Case XIII.—*Stenosis due to cicatrices (?) following intubation for laryngeal diphtheria and to the cicatrices following improperly located tracheotomies; apparent cure followed by contraction of the cicatrices; necessity of indefinite tubage; still under treatment.*

G. R. was admitted to the Willard Parker Hospital at the age of one year and intubated on December 17, 1900, on account of laryngeal diphtheria. The stenosis persisted, and therefore the tube was retained until March 28, 1901, when respiration, though slightly difficult, was possible without assistance. He was sent home soon afterward, but the dyspnœa increased, and on April 18th he was brought back to the hospital and again intubated. The stenosis persisted and the tube with frequent changes was retained until November 1st. Then, as respiration was possible without assistance, he was taken home; but the stenosis gradually reappeared and later he was brought to the Beth Israel Hospital, and in the first part of December, 1901, tracheotomized.

On January 3, 1902, he was admitted to Gouverneur Hospital, where I found that though a small amount of air passed through the larynx, respiration without the tracheal cannula was impossible. The fistula was just below the cricoid and entered the trachea through its lateral aspect, a position which seems peculiarly favorable to the subsequent development of stenosis. The anterior large segment of the cut tracheal rings are inverted by the cannula and the dividend extremities are thus very prone to override. Under chloroform a six-to-seven intubation tube was introduced easily through the larynx, but only passed the fistula with the use of some force. At the end of a month, as respiration was only possible for a short time, a large special rubber tube of the six-to-seven length, but two thirty-seconds of an inch larger in the transverse diameter at the neck and retaining swell, was employed.

On April 30, 1902, the tube was removed, and a week later the patient discharged apparently cured.

Readmitted to Gouverneur Hospital on November 27, 1903, with marked dyspnœa, which had only appeared a few days previously. Attempts with intubation, even with the smallest tubes, failed, the resistance being opposite the tracheotomy cicatrix, and asphyxia was barely escaped by another tracheotomy. Through this opening the stenosed trachea was rapidly dilated up and down with urethral sounds until the large special tubes previously employed could be inserted. This was worn with infrequent changes for nearly two months, and then, as all dyspnœa seemed relieved, a normal six-to-seven tube was inserted for a week to guard against possible adductor spasm. This was removed and on February 27, 1904, the child was sent home apparently cured. But on March 8, 1904, he returned with marked dyspnœa. Intubation was again pre-

vented by the tracheal cicatrix and another hasty tracheotomy followed by dilatation was required. The large special tube was again used, changing and cleaning every four or five weeks, and on June 10, 1904, it was left out and the child dismissed. But on July 2d the parents having noted that the breathing was becoming noisy, brought him back, and he was again intubated with the large tube. On November 1, 1904, he was dismissed without the tube, only to return showing slight dyspnœa on November 26th, and was reintubated as a matter of precaution. The wearing of this instrument is perfectly safe at home in this case, as without it for a week or more there is no difficulty in breathing, and with it the child is in all outward respects normal, except that the voice, of course, is only a whisper.

The tube, which is about the calibre of this child's (normal) trachea, is still worn and will be continued with the usual changes for cleansing for a year in hopes that, as in some of the other cases, the cicatrix will eventually cease to contract. Excision of the cicatrix and accurate coaptation of the cut anterior ends of the tracheal cartilages is the remaining alternative. But as the cicatricial tissue occupies nearly the whole front of the cervical trachea there would be considerable risk of failure to obtain primary union, and the result of such an operation might be worse than the present condition. It should only be undertaken as a last resort. In the meantime this child goes to school and is to all outward appearances as normal as his playmates, with the exception of his voice.

Since the last note the tube has been changed once a month and no dyspnœa has been apparent and no sign of recontraction. But after the experience of the previous recontractions it is my intention not to attempt permanent removal of the tube until next December. A year of uninterrupted dilatation should produce a cure, and if it does, will be valuable from a prognostic standpoint in other intractable cases.

Case XIV.—*Stenosis due to cicatrices following intubations for laryngeal diphtheria; cured by prolonged dilatations after six months.*

[The following history is supplied to me by Dr. F. L. Taylor, who had charge of the patient, and has kindly consented to the report.]

A. K., aged 3 years, was hastily intubated by Dr. Taylor with a six-to-seven tube (one which had been prepared for another case and was too large) on November 20, 1903, when practically moribund from asphyxia due to laryngeal diphtheria. A week later the tube became clogged and was replaced by one of the proper size for the child. Repeated attempts at permanent removal during the next six weeks were followed by gradually recurring dyspnœa. This dyspnœa came on within a few hours after extubation at first (the œdema of an ulcer) and later within a few days (the stenosis of a contracting cicatrix), and on January 3, 1904, some resistance to the insertion of the tube was noted.

A "special" tube was inserted on February 17th, having the diameter of the neck three thirty-seconds of an inch smaller than the retaining swell, which was as large as that of a normal six-to-seven tube, the largest which by experiment could pass the larynx without undue force. This was several times coughed out during the next week, and some adductor spasm was noted, so a three-year tube was inserted. Its re-

moval a few days later was followed by a gradual recurrence of the dyspnœa, and on March 26th, as the special tube was coughed out, a six-to-seven tube, or the largest normal one which could enter, was inserted. This was worn until May 23d, and then for a week the normal three-year tube was worn. There was no recurrence of dyspnœa thereafter, and the patient is now (October, 1905) in all respects normal.

This is only a mild case of chronic stenosis, but I have selected it from many similar histories in my possession to illustrate the dangers of traumatism in acute diphtheria (unavoidable in this instance) and the proper treatment of the subsequent cicatrix. Hypertrophic laryngitis did not occur.

Case XV.—*Stenosis from cicatrices of syphilitic origin; clogging of a metal intubation tube; extreme subsequent contraction of the larynx which required laryngotomy to insert a plugged tube; apparent cure followed by apparent failure owing to unrecognized spasm of the adductors.*

C. McD., aged 25 years, was admitted to Gouverneur Hospital August 18, 1896, suffering from marked laryngeal dyspnœa, which had been increasing gradually since the previous April. There was an indistinct history of syphilis. She was immediately tracheotomized and, as is usual in emergency cases, the cricoid cartilage was divided. Subsequent examination revealed a larynx much deformed and obstructed by cicatrices. There was no ulceration. On November 15, 1898, under chloroform the larynx was dilated up to No. 35 F., with urethral sounds entered through the trachea and a metal tube inserted. Much difficulty in swallowing followed and a week later I arrived just in time to extract the tube, which had become nearly occluded by dried mucus. Another intubation was refused and the tracheal cannula had to be inserted. Attempts to use a Dupuis' segmented right-angled cannula failed, owing to the difficulty in swallowing and to the pain and swelling in the top of the larynx over the upper end of the superior half of the instrument. There was the same difficulty with an ordinary curved cannula, to which was fitted on its upper surface a bivalve dilating attachment to extend up into the larynx. Intermittent dilatation with Schroetter's tubes proved useless, as is usual in these bad cases. The patient left wearing a tracheal cannula. In February, 1899, she returned and under chloroform the larynx was found almost but not quite occluded. The thyroid and remains of the cricoid cartilage were then split in the median line upward from the cicatrix and the tortuous line of mucuous membrane representing the remains of the lumen of the larynx was laboriously followed with a fine probe, as in external urethrotomy without a guide, until the pharynx was opened. A small adult intubation tube was then inserted through the mouth, and after making longitudinal incisions in the most prominent cicatrices the wound was sutured over the tube and a tracheal cannula placed in the lower angle beside the tube. A couple of weeks later an intubation tube with its anterior half cut away to receive the inner end of the cannula was attempted, but clogged with dried mucus and pus so often that it was abandoned and a "plugged" tube was substituted. This worked fairly well, but the fistula in healing came to be located about the middle of the thyroid cartilage. It, however, brought the tube near

the surface and the patient could clean it daily with a feather after removing the plug. A change of the tube every four or five weeks demonstrated rapid improvement, and in May the tube was removed; the fistula in the neck contracted almost immediately, so that at the end of twenty-four hours it was practically closed. Meanwhile natural respiration was carried on without the slightest difficulty, but suddenly three or four days later, at night, an attack of hyspnœa almost caused asphxia (adductor spasm), but fortunately enough of the fistula remained to enable the house surgeon to dilate it and insert the cannula. Further treatment was then refused for some time and the patient went home wearing the tracheal cannula. April, 1900, under chloroform, the contracted larynx and trachea were dilated as before. But so much difficulty in intubating was experienced on account of the "spur" which had formed in the posterior wall of the trachea opposite the fistula, that the attempt had to be postponed until a small right-angled blunt retractor, grooved on its convexity to fit the intubation tube, could be constructed. A week later, under chloroform, with this instrument in the lower angle of the fistula to drag the trachea forward and to guide the tube in the convexity of the retractor into it, intubation was accomplished. The tube used was one with a "plug" attachment. This was kept in place with occasional changes for cleansing for about four months or until August, 1900, then a cure seemed obtained, but two days later, the fistula having nearly closed, there was a sudden attack of dyspnœa as before at night and the cannula was reinserted and the patient refused further treatment. When seen last about a year later she was pregnant and showed signs of advanced pulmonary phthisis and has probably died since then, but at that time, though the cannula was still worn, laryngeal respiration, much to my surprise, was fairly good and another course of intubation would probably have produced a cure. But it seemed hardly justifiable in her condition. The apparent lack of success after prolonged dilatation was undoubtedly due to my failure to recognize and allow for the "cramp" of the laryngeal adductors. A tube with a small neck worn for a couple of weeks after the removal of the large ones would have permitted the adductors to regain their strength and so prevented the cramp or spasm of the adductors. She was a a very nervous woman, somewhat addicted to beer and easily terrified, so that the sudden dyspnœa after apparent success would make her refuse trust in anything but the cannula. In the light of later experience I have regarded this case as really a success, though apparently a failure.

Case XVI.—*Stenosis due to cicatrices of syphilitic origin; difficulty in swallowing with a "clamped" tube; cured by a primary dilatation with urtethral sounds and by wearing a "clamped" tube for ten days in February and for thirteen days in July.*

P. M., aged 32 years, had syphilis eight years ago and for the past six months has had gradually increasing laryngeal dyspnœa. He was first seen on January 16, 1903, and was then nearly asphyxiated from an acute laryngitis grafted upon the pre-existing chronic difficulty. A hasty tracheotomy was performed and soon after inunctions of mercury instituted without improvement of the stenosis. On February 1st, the acute

conditions having subsided, examination revealed that only a stump of the epiglottis remained and that the larynx was nearly completely occluded with a mass of irregular cicatrices; but there was apparently no area not covered by epithelium and no sign of inflammation. By closing the tracheal fistula it was found that respiration, though very difficult, was just possible. Though it seemed hardly credible, the patient said it was about as bad for several weeks previous to the tracheotomy. Under chloroform at St. Francis Hospital the larynx was rapidly dilated with urethral sounds, beginning with No. 10 F. and ending with No. 88 F. A very considerable amount of force was used and some tearing of dense fibrous tissue was perceptible. A large adult intubation tube with a "clamp" attachment was then inserted with some difficulty.

On February 9th the tube had to be removed, as the patient had scarcely swallowed a mouthful of food or water since being intubated. Fear of choking seemed to have more to do with the difficulty than pain, of which there was no complaint after the first three or four days. Respiration, though slightly noisy, was excellent compared to the period preceding the tracheotomy, but that experience had so terrified him that he insisted on wearing the tracheal cannula. He refused further treatment and went home until July 10th, when he re-entered the hospital. Considerable contraction had occurred in the larynx and only the merest trifle of air could pass the stenosed region. There was a marked spur formation in the posterior tracheal wall opposite the fistula. Under chloroform the larynx and upper end of the trachea were quickly dilated with sounds entered as before through the fistula and the large adult tube with the clamp attachment inserted. The same difficulty with swallowing followed as before, and much against my will I was compelled to remove the tube on July 23d. But there was no return of the hyspnœa: the fistula quickly closed, and at present, with the exception of a husky voice, the man has done well. The "mixed treatment," which was taken more or less irregularly, seemed to have had no effect on the cicatrices.

There was no sign of obstruction in December, 1904. The difficulty in swallowing while intubated experienced by this patient may have been partly due to the absence of epiglottis, which had been almost completely destroyed by the syphilitic process before he came under observation.

Case XVII.—*Stenosis due to cicatrices of syphilitic origin; unusually rapid and permanent recovery after dilatation and intubation; duration of treatment only eight days.*

M. C., aged 45 years, was admitted to Gouverneur Hospital December 16, 1898, with an acute exacerbation of a long-standing laryngeal stenosis apparently of syphilitic origin. A hasty tracheotomy was needed and the cricoid and one or two rings of the trachea were cut well to one side of the median line. Considerable infection of the neck followed, but subsided. Examination revealed a larynx with its lumen much deformed and nearly obliterated by cicatrices. There were no ulcerations.

Under chloroform on December 27th the larynx was rapidly dilated with urethral sounds entered through the fistula up to No. 40 F. Some fibrous tearing was perceptible. A normal large adult tube was then inserted. Great difficulty in swallowing resulted and scarcely a mouthful

of food or water could be taken until January 5, 1899, when the tube was coughed out. As no hyspnœa followed, it was not reinserted and the man left the hospital shortly afterward. This patient was shown at the Surgical Section of the Academy nearly two years later and no return of the stenosis had occurred, though the voice was husky. He received "mixed treatment" only during the period from December 16th to 27th, and on the 27th no improvement of the cicatricial condition was apparent as judged by the resistance to the passage through the strictured region of the urethral sounds. This was the most rapid and permanent recovery of one of the worst cases in the series.

Case XVIII.—*Stenosis due to post-typhoidal perichondritis and also to cicatrices and deformities following tracheotomy; under treatment with "plugged" and clamped tubes; marked improvement; still under treatment.*

J. P., aged 25 years, had a very severe attack of typhoid fever in July, 1900, during which he was delirious. Laryngeal dyspnœa occurred and he was only saved from asphyxia by a hasty tracheotomy. This was probably a typhoidal perichondritis. He eventually recovered, but never afterward could breathe without the cannula.

On October 7, 1904, Dr. Delavan, who kindly invited me to be present, found at this time an ankylosis of the left arytenoid cartilage holding the corresponding vocal cord in the median line; the tracheal fistula was just below the cricoid cartilage which was tipped downward in front; the trachea was contracted around the cannula to about the size of a No. 20 F. sound; there was a marked "spur" opposite the fistula in the poster or tracheal wall; the upward opening into the larynx was slightly smaller. Respiration was fairly comfortable in repose, but at night the dyspnœa necessitated the constant wearing of the cannula and the slightest exertion made laryngeal breathing difficult. Under cocaine I made attempts to dilate this stricture, which was worse around the fistula, by passing sounds through it up and down. The tearing of the fibres in the cicatrix could be distinctly felt, and I at length had to desist without intubating for fear of rupturing into the œsophagus. The reaction following these somewhat violent manipulations was considerable. There was a great deal of swelling, some purulent discharge, and fever and respiration through the cannula was continued until this subsided on November 1st.

Then after the usual dilatation a small adult rubber tube was inserted with great difficulty. This was caused chiefly by the "spur" opposite the fistula, which could not be passed by the lower end of the intubation tube until a small special retractor, grooved on its convexity to guide and push back the tube, was inserted into the lower angle of the tracheal fistula to pull it forward. The "spur" made the tip of the tube catch in the tracheal fistula and without the retractor or without widely opening the trachea, which is very undesirable. I have found this difficulty quite troublesome.

A few hours after intubating dyspnœa began and soon became urgent. Examination revealed that swelling had occurred chiefly in the epiglottis and glossoepiglottic folds, which caused these structures to overtop and

nearly occlude the upper opening of the tube. Extubation and insertion of the cannula relieved all symptoms. A week later the swelling had subsided and respiration through the larynx was found much improved and sounds up to No. 39 F. were passed through the fistula.

On November 16th, under cocaine, the larynx was dilated up to No. 42 F. and a large adult normal tube inserted with considerable force. On November 21st a large "plugged" tube was substituted for the normal tube to avoid any possibility of autoextubation. Laryngeal respiration was found to be perfect, but with so short a dilatation recontraction was certain, and therefore the large plugged tube was left in place until December 16th.

Removal of the tube on this date revealed no abnormality, except, of course, the tracheal fistula, which had been purposely kept open by the plug. The laryngoscope showed that even free mobility had been restored to the left arytenoid, which previously had been fixed and apparently ankylosed. The relief from pressure caused some slight swelling during the first few days, but never enough to interfere with laryngeal respiration. A straight cannula closed at its outer end by a cork was retained in the fistula in case of any recontraction. (This cannula, by the way, is only a quarter of an inch in diameter and simply maintains the potency of the fistula until the danger of adductor spasm or sudden œdema is past.)

Although respiration was free, trial of the larynx on December 20th, with the tube recently removed, showed some resistance, and therefore a very large clamped tube was inserted and the patient sent home to California. This tube became foul, and on February 14, 1905, the patient *extubated himself*. At first respiration was normal, but a few days later he noticed some dyspnœa on exertion and was reintubated with a slightly smaller tube. This met with some resistance, and so demonstrated recontraction and the necessity of a continuation of the treatment. Since then he writes me that he has learned not only to extubate but to *intubate himself*, an accomplishment which I should not have thought possible. I have advised that the intubation be continued uninterruptedly for at least six months. This patient has recently written me (October, 1905) from his home in California that he has been comfortably breathing for two weeks without his tube, but as he noted some stridulous breathing, he was about to reinsert the instrument. It remains to be determined how long a time interrupted dilatation may be required to effect a cure. The temptation to remove the tube is obvious in a case like this.

Case XIX.—*Stenosis due to post-typhoidal perichondritis complicated by the cicatrices and deformities following a tracheotomy; cured by four months' dilatation with a normal hard-rubber tube.*

[The following two histories are of patients of Dr. Emil Mayer. As I had the honor of occasionally advising in their progress and as they present much of interest, I have included them here with Dr. Mayer's permission.]

H. H.,* aged 28 years, passed through a severe attack of typhoid fever in October to December, 1902. On January 13, 1903, a gradually increasing dyspnœa finally required a tracheotomy by Dr. Mayer. Later interrupted dilatation with Schroetter's tubes failed to enable the patient to breathe without the cannula. From time to time there were noted general swelling of the soft parts above the cords, adhesions between the posterior extremities of the cords, fixation of the left half of the larynx, and granulations about the fistula. Eventually there developed a cicatricial stricture at and above the fistula and some displacement forward of the trachea below. On April 20th under chloroform this was rapidly dilated with urethral sounds passed up and down through the fistula and a No. 7 adult rubber intubation tube inserted by me. The fistula was allowed to close and the tube changed June 8th. The patient then went abroad prepared with a full set of instruments for the emergency of autoextubation. No difficulty arose, and on her return on September 1st the tube was removed. Twenty-four hours later, as the larynx had become œdematous and dyspnœa quite marked, the largest child's tube was inserted by Dr. Mayer. This was coughed out on September 4th. Examination then revealed swelling and fixation of the left half of the larynx and some granulations below the anterior end of the cords and some narrowing and thickening of the larynx at the level of the cricoid cartilage. The largest child's metal tube was inserted September 10th and left for ten days. Since its removal there has been no recurrence of dyspnœa. The only abnormality at present is a voice which is rather husky and deep-toned.

Case XX.—*Stenosis due to cicatrices following a compound fracture of the laryngeal cartilages; dysphagia with a plugged tube; failure to cure.*

J. M.,† aged 25 years, was admitted to the New York Eye and Ear Infirmary December 5, 1898, under the care of Dr. Mayer. A Mauser bullet at El Caney, June 2, 1898, had passed obliquely through the right temporal region and superior maxilla, shattered the thyroid cartilage and then penetrated the pharynx, apparently entering the stomach through the œsophagus. Though almost unable to swallow, he survived this wound, starvation, and extreme dyspnœa, which was for some unknown reason allowed to persist, until he was tracheotomized upon arrival at Bellevue Hospital on August 3d.

Respiration was thereafter only posible through the cannula. After being transferred to the Eye and Ear Infirmary Dr. Mayer found the lumen of the larynx completely occluded by cicatrices, which seemed everywhere covered by mucous membrane. After persistent efforts a Schroetter dilator was finally passed, and in course of time the stricture was dilated enough to admit a dilator of considerable calibre. But after its withdrawal the larynx collapsed each time and no air could pass it. On March 7th, 1899, after repeated trials a large rubber intubation tube was inserted. The patient seemed unable to swallow with this in place and every few days it was coughed out. Hence, at my suggestion, on April 24th a large "plugged" intubation tube was substituted. The patient was even more

* Proceedings of the American Laryngological Association, June 2 to 4, 1904.

† Reported to Section on Laryngology and Otology, Fifty-first Meeting of the American Medical Association, June 5 to 8, 1900.

unable to swallow with this apparatus than with the other and had to be fed by a stomach tube, which he finally learned to introduce himself. On July 20th the intubation tube was removed, and as the larynx was entirely open and even the voice good, it was not inserted and the fistula was allowed to heal. Ten days later as there seemed some swelling in the larynx, though there was no obstruction, some local application was made and immediately followed by a sudden dyspnœa, which forced Dr. Mayer to dilate the nearly closed fistula and reinsert the cannula. After five days, or on August 1, the plugged intubation tube was put back, but a cellulitis of the neck developed, and this, with the difficulty of swallowing, so reduced the patient that, although a little more perseverance seemed likely to produce a cure, he eventually preferred a pension to further treatment. When last seen by Dr. Mayer in June, 1900, there was some deformity of the interior of the larynx and evident narrowing of the space within the cricoid cartilage. But the vocal cords were visible and functionated, and, although the cannula had to be constantly worn, a considerable amount of air passed through the larynx.

Case XXI.—*Stenosis due to an intralaryngeal tumor; thyrotomy; infection of the wound and bad cicatricial contraction and deformity of the larynx and trachea; rapid dilatation, followed by the prolonged use of a very large clamped "special" tube which overdistends the whole cicatricial area; persistent fistula which was excised together with much cicatricial tissue; appearance of papillomata and their excision with apparent cure.*

B. W., aged 3 years, a patient of Dr. Harris, came to him with a history of laryngeal dyspnœa gradually increasing for several months. For this Dr. Harris performed a thyrotomy and removed an angiofibroma from within the anterior portion of the larynx on August 10, 1904. The wound suppurated and the larynx was packed, leaving the cricoid cartilage partially exposed and undivided and the cannula in the tracheal fistula just below it. On September 28, the wound having healed and the larynx being so contracted that no air could pass through it, under chloroform, with Dr. Harris' assistance, I rapidly dilated the larynx with urethral sounds, beginning with a No. 5 F. and continuing up to No. 28 F. These were entered through the fistula. The stricture seemed to occupy, as might be expected, nearly the whole laryngeal space from the fistula up, and though evidently fibrous, was not very resistant. A large special tube of the four-to-five length was inserted. Considerable reaction followed; the temperature rose to 104 degrees the next day, and some purulent discharge showed through the fistula, but this gradually subsided, and on October 14 the tube was coughed out, nearly causing asphyxia. The house surgeon failed to intubate, and so reinserted the tracheal cannula by dilating the nearly closed fistula. As the child was in poor general condition, this was left until November 14, and then with cocaine application locally the larynx was found to have become not only stenosed but deformed. There was also some "spur" formation posteriorly opposite the fistula. After many attempts Dr. Harris finally managed to pass the smallest sound he had (a No. 5 F.) from the fistula into the pharynx. The stricture was then rapidly dilated up to No. 25 F. and a large special clamped tube of the four-to-five length inserted. The manipulations

with intervals for breathing occupied over an hour and some reaction in temperature followed, but no pneumonia could be detected. On November 24 the clamped tube became foul and was removed. There was no difficulty in breathing with the tracheal fistula occluded, but another very large clamped tube was inserted to overdistend the extensive cicatrix.

This large clamped tube was removed on January 1, 1905, and a normal tube substituted to test the amount of contraction which would take place. Ten days later the large tube was found to enter the larynx without undue force; in other words, there seemed to be no tendency to recontraction, but to make sure, the large clamped tube was left in place until February 1. On this date, as respiration was free and the fistula in the neck present in case of adductor spasm, the tube was removed. A few days afterward some "spasm" occurred during the night and the house surgeon inserted a normal tube without difficulty. The cervical fistula had contracted to a very small opening. A few days later a sharp attack of pneumonia developed and I removed the tube with the idea of leaving the child in as natural a condition as possible to combat the disease. Although I have several times observed a pneumonia in these tube cases run a perfectly normal course unaffected by the tube, I have always felt that an unobstructed larynx was preferable to one containing a foreign body. Hence, as there proved to be no obstruction in this larynx, the tube was not replaced. At the end of a week the pneumonia subsided and a slow convalescence ensued.

The house surgeon on February 25 noted some noisy respiration while the child was sleeping, and, fearing a sudden "spasm," inserted a normal tube. Three days later a small pustule appeared near the upper end of the cicatrix over the thyroid cartilage, and after evacuating a drop or two of thin pus I removed the tube again. There was no difficulty in respiration. There had apparently been some infection through the pharynx or larynx and the consequent œdema had seemed to require the tube. As no recontraction nor dyspnœa followed and the fistula into the trachea in the lower part of the cicatrix had become lined with epithelium, on March 8 under chloroform I excised the fistulous tract and the surrounding cicatricial tissue up to the cricoid cartilage, and exposed and brought into accurate coaptation over a special tube the cut anterior ends of the tracheal rings. No ulcerations were visible within the larynx or trachea. Four days afterward the superficial parts of the wound broke down, but as the wound in the trachea and overlying muscles held together, there was some hope that the stenosis apparently cured before the operation might be permanent. About the middle of April some fever was noticeable, but immediately subsided after changing and cleansing the tube.

On May 27th a laryngoscopic examination was made under chloroform by Dr. Harris, and, as no sign of obstruction could be detected, the tube was left out and shortly afterward the child was sent home. In August she reappeared with bad inspiratory but no expiratory dyspnœa, and examination with the finger revealed what I thought were "granulations" around the upper aperture of the larynx. Under chloroform I scraped these away and inserted a metal tube with the head built up to protrude above and overlap the supposed ulcerated area. After returning from a vacation a month later I found the "granulations" worse than

ever, and then with Dr. Harris' assistance discovered that the new tissue consisted of papillomata.

On October 3, 1905, the larynx and upper trachea were again opened in the median line and all the new-growth trimmed off with scissors and the raw surface touched with nitrate of silver. The widely opened larynx appeared otherwise normal, except that the first operation by Dr. Harris had necessitated removal of the greater part of the vocal cords and these structures had not reformed. The wound was closed tight with catgut, and though the natural respiration demonstrated the complete removal of the growth, a normal intubation tube was inserted to guard against œdema. Primary union followed and the tube was taken out on October 12th. There is no sign of obstruction as yet (two weeks later), and it remains to be seen whether the papillomata will recur.

Case XXII.—*Tuberculous (?) or traumatic laryngitis which in healing occluded the larynx; cured (?) after seven months' dilatation.*

M. C., aged 38 years, began in June, 1901, to suffer from "hoarseness," which was evidently regarded by her physician as due to tuberculous laryngitis. The larynx was several times "cauterized," but in spite of climatic and other treatment pain, dysphagia, and dyspnœa finally developed, and on November 1, 1902, she was intubated with the ten-to twelve child's tube for a short time. After its removal the dyspnœa recurred, and on January 11, 1903, she was tracheotomized at the New York Hospital and afterward could not breathe without the cannula. She then came under Dr. Delavan's care and with his assistance and advice the present treatment was inaugurated. With the greatest effort it was found that enough air could be forced through the larynx to make articulation barely intelligible, though examination revealed no aperture, the larynx apparently being occluded at and below the vocal cords. The cervical fistula was large and through the second and third tracheal rings a little to the left of the median line and covered with epithelium throughout.

Under chloroform, on March 22, 1905, the smallest urethral sound could not be made to pass the stricture either from below or above, but by working from below through the fistula a fine probe was finally passed up into the pharynx. This was followed first by a fine dilating forceps and then by urethral sounds of increasing calibre in rapid succession entered through the fistula, until No. 40 F. was admitted with considerable resistance. The stricture seemed to be of the nature of a thick diaphragm at and below the vocal cords. Strangely enough, there was no contraction or deformity around the fistula and no troublesome "spur" opposite it posteriorly.

A No. 6 adult intubation tube was then inserted without difficulty and worn with very little complaint until March 29th, or one week. After its removal the larynx, including the cords, seemed normal, except for some ulceration, especially under and involving the right cord. She was inspected daily, and as the ulceration was very close to the anterior commissure, and as a band seemed to be developing at that point, on April 3d the No. 6 tube was reinserted without difficulty to be worn until this ulceration could have healed. On May 8th the tube was again removed and

everything found normal; the mobility of the cords was excellent, but they were deeply congested. A small whitish protuberance existed below the anterior commissure. This seems to be the remnant of the original cicatricial diaphragm, but there is no obstruction to respiration. A week later as these protuberances had slightly enlarged, although there was no obstruction to respiration, it seemed wise to reinsert the tube in hopes they might ultimately disappear. Another change of tube on June 5th showed that the larynx was freely open. But after four days Dr. Delavan noted that the former cicatrix just below the anterior commissure of the cords was again becoming apparent, and although the patient was unconscious of any difficulty in respiration, it seemed wise to replace the tube and continue the dilatation. The tube was again removed August 1st for two weeks and then replaced on August 15th. On October 4th it was taken out again, and as after three weeks no recontraction is apparent, a cure is hoped for, but cannot yet be really expected.

Nearly all of the cases described above have been presented before one or more societies, but the few other histories I could submit are withheld, as they have been lost or are too incomplete to be of value. I should, however, mention one case of (presumably) syphilitic stenosis of the larynx seen with Dr. W. K. Simpson, if only to avoid the charge of suppressing an unsuccessful treatment. She was a woman, aged 35 years, who had worn a tracheal cannula for a couple of years. Under ether the stricture was dilated as usual with urethral sounds and a large tube inserted. This has been worn for over two years, and at present the patient is unwilling to submit to its removal. In the first part of this period one or two attempts were made to dispense with the tube, but each time a recurrence of the dyspnœa compelled a reinsertion of the instrument; hence at present I must report her in one sense as a failure, although in another her condition is infinitely preferable to the deformity and danger accompanying the permanent use of a tracheal cannula. She is to all outward appearances, with the single exception of her voice, which is, of course, only a whisper, a normal woman in good health.

All the credit for the main principle in the management of chronic cicatricial laryngeal stenosis, that of long-continued dilatation with an intubation tube, of course belongs entirely to the late Dr. O'Dwyer. He long since insisted that this method was the only one generally applicable, and he published several cases of chronic stenosis due to one or another cause which were brought to a satisfactory conclusion by intubation continued for several months. But he had no regular course of procedure for curing the occasional cases of "retained tube" with chronic hyper-

trophic laryngitis, which in addition are often complicated by dangerous auto extubation, nor did he go into the details of the management of the various conditions accompanying stenosis with a retained tracheal cannula, and in these small particulars the histories of the cases given above supply much that is valuable. For there is no more serious disability than chronic respiratory obstruction in the upper air passages, and any method of treatment which is safe and offers a reasonable prospect of cure in nearly every instance should be welcome. Great patience, perseverance and foresight with some technical skill are required, but given these my experience has led me to believe that a failure to ultimately restore natural respiration and even voice must be exceptional. Intermittent dilatation with Schroetter's instruments and all the various operative procedures which from time to time have been recommended chiefly in German literature are notoriously unsatisfactory. Some conditions must, however, occasionally be encountered which can only be remedied by plastic or other more or less extensive surgical interference, but they are rare and even then subsequent cicatricial contraction will probably arise. Primary union in the several laryngotomies mentioned above was much less common than suppuration, which may vary from simple failure of the wound to unite by first intention to extensive cellulitis of the neck with lymphadenitis and sloughing of the cartilages. In short, operations for stenosis of the larynx or upper trachea are seldom advisable except for the removal of tumors. Papillomata are probably the most common of these, and several cases have been reported in which intubation alone was said to have caused their disappearance. But without the definite knowledge of the extent of the disease only to be gained by a previous thyrotomy or tracheotomy intubation may prove dangerous; the papilloma or any other growth may be caught in the point of the tube and masses of this tissue pushed down ahead of it into the trachea like a gunwad. Several years ago I encountered a death from asphyxia in the practice of a colleague produced in this way. Hence, of course, intubation, like any other surgical procedure, cannot be indiscriminately employed, and an open operation, if only to make sure of the conditions, may sometimes be a necessary preliminary. But aside from the inspection or removal of new growths the causes of stenosis rarely require anything beyond dilatation followed by intubation.

These cases can therefore generally be grouped as obstruction due to (*a*) cicatrices, (*b*) hypertrophic laryngitis, (*c*) granula-

tions or œdematous ulcer, and (*d*) spasm of the adductors. One or more of these conditions may coexist and before proceeding to the treatment it is necessary to differentiate briefly the symptomatology. A cicatrix is easily recognizable by the history of a traumatism or disease with or without ulceration, coupled with inspection and instrumentation of the larynx, or tracheal fistula if one has been made. The deformities common after tracheotomy have already been described. The chief difficulty lies in differentiating between some of the tumors, including the granulomata and the persistent stenosis—hypertrophic laryngitis—sometimes following intubation for an acute condition, generally laryngeal diphtheria. The tumors, of course, if they cannot be removed through the mouth, require open operation anyway, and exploration is generally safer than working in the dark. But the differentiation of a cicatrix from the other common conditions should be simple even when an open ulcer overlies it. In the first place it offers considerable resistance to instrumentation, and after dilatation or intubation the stenosis returns gradually within days or weeks. An hypertrophic laryngitis offers no resistance to the insertion of a tube, and after its withdrawal the recurrence of the obstruction is immediate and often complete. When these two difficulties occur together, as is not infrequent, especially after diphtheria, the history coupled with the resistance to instrumentation and the sudden recurrence of dyspnœa after it, should present no great difficulties, although any traumatism of the larynx may give rise to an œdema of quite sudden onset. The spasm of the adductors, though not strictly speaking a cause of chronic stenosis, so closely simulates clinically an hypertrophic laryngitis that it must be included in the differentiation. It seems to have been recognized long before the days of intubation and so must occur whenever the laryngeal muscles have been for any length of time at rest. Of course, when an intubation tube has been worn for a considerable period, particularly the "special" instrument which I recommend with an abnormally large neck, the adductors are put out of employment and must undergo an atrophy of disuse while the adductors are under some more or less constant tension with every attempt at coughing. However the explanation may be made, clinically at least there is no difference appreciable between spasm of the adductors and hypertrophic laryngitis after removing the intubation tube or plugging the tracheal fistula. When, however, this condition is suspected in a patient who has been intubated the diagnosis can sometimes

at least be made by extubating under an anæsthetic. This removes the element of fright and abolishes the disturbances somewhat vaguely termed "reflexes," and normal respiration may be discovered where it might not otherwise be anticipated. Furthermore, a sudden spasm, and that, too, of very dangerous dyspnœa, generally occurring at night with coughing, and inspiratory rather than expiratory, can pretty safely be ascribed to this cause. The resistance encountered in this condition by the tip of an intubation tube catching on the closely approximated vocal cords should not be misinterpreted as a cicatrix, which takes time to develop and contract. The treatment of the two conditions is very different. "Granulations," if not perceptible to sight or touch, bleed easily upon instrumentation and generally occur as the result of the pressure or irritation of an intubation tube or cannula, in which they produce a more or less gradually increasing obstruction either to inspiration or expiration, seldom to both acts in succession. A spasmodic cough accompanied by more or less hemorrhage and dyspnœa may also give some indication of their presence; but I am convinced that they are a far less frequent cause of chronic obstruction than seems generally to be believed. When there is bleeding accompanied by stenosis following extubation or decannulization it is much more reasonable to ascribe the dyspnœa to a simple œdematous ulcer overlying an hypertrophic laryngitis or a cicatrix and not to exuberant granulations. The slight traumatism incident to the relief of the pressure of an intubation tube against such an ulcer or the "gagging" which often accompanies the removal of a cannula, especially if it is high up, can produce enough congestion in an eroded area to cause it almost immediately to nearly or quite occlude an already narrow space. I have never been able to positively verify such a condition but believe in its occurrence, and though it may be impossible to differentiate from a pure hypertrophic laryngitis, which is also very vascular and bleeds at the slightest touch, the treatment does not differ.

In a former article on this subject I recommended the long-continued use of as large a normal intubation tube as the larynx would admit, but experience has caused me to modify this treatment very materially for the different conditions. Simple chronic hypertrophic subglottic laryngitis is probably the most common cause of obstruction after intubation. This ought to read "after diphtheria," because it seems unfair to imply that it is due to intubation inasmuch as it was recognized as one of the most fre-

quent obstacles to the removal of a tracheal cannula long before intubation was invented.* It may also occur entirely independently of any acute disease, and Case I. seems to be an example of this. There will, however, be considerable difficulty in convincing most intubators that unskilled or rough instrumentation does not have something to do with its inception or continuance, for a certain number of these cases will get well in the course of days or weeks with careful and frequent change of a tube, smaller if need be than the development of the patient would ordinarily indicate, the object being to minimize traumatism. Local astringents, as advised by Dr. O'Dwyer, applied to the interior of the larynx in a coating of gelatin or other substance adhering to the outer surface of the tube, may also succeed. But there are a number of cases (about 1 in every 100 intubated as estimated from the statistics given in my previous paper†) which will resist all these efforts for their relief. If the stenosis of this character has persisted for about a month in spite of all the usual plans of treatment, and spasm of the adductors can be excluded, another method must be adopted. Some of the patients will recover if as large a normal tube as can pass the cricoid cartilage is used for five to six months and changed at intervals only for cleansing. At the end of every four or five weeks the tube becomes foul and causes a bad odor in the breath and considerable cough. But usually in a persistent case it is better to start with a "special" tube or one having the neck one or more thirty-seconds of an inch larger in the transverse diameter than the normal instrument, with the object of applying pressure upon the thickened area within the larynx. As this area generally extends from above the vocal cords to or through the cricoid cartilage, and as the retaining swell of the tube must be enlarged with the "neck" to prevent autoextubation the retaining swell of the special tube must be enlarged along with the neck; and the same must be done with the head or there is danger of the instrument slipping below the epiglottis, which will then more or less completely occlude the upper aperture. Practically in these cases I am accustomed to make trials with normal tubes of increasing calibre under complete anæsthesia if necessary, until I have found the largest tube which will enter and pass through the larynx with some slight force. The "special" tube is then made of the length of the normal tube proper for the age and development of the patient, but

* Köhl. Archiv f. klin. Chir., 1887, Band XXXV., p. 75.

† Annals of Surgery, May, 1905.

with a retaining swell having the diameter of that of the trial tube. The neck must then be at least three thirty-seconds of an inch smaller than the retaining swell and the head six thirty-seconds of an inch larger than the retaining swell. If a metal tube is to be used its weight is better and more safely sustained by a head which is at least seven thirty-seconds of an inch larger than the retaining swell and a large tube should have eight thirty-seconds of an inch difference. If the child is under four or five years of age and so needs the smaller sized tubes the thirty-seconds of an inch difference should be altered to sixty-fourths of an inch. Moreover, a difference of three thirty-seconds of an inch between the neck and retaining swell is the minimum difference which I have found by experience sufficient to keep the tube in place. Some patients will require four thirty-seconds or even five thirty-seconds of an inch difference. In the first part of the treatment of an hypertrophic laryngitis, especially if the stenosis is complete, as is frequently the case, it is wise to start with a special tube having a diameter of the neck five thirty-seconds of an inch less than that of the retaining swell. Care in these seemingly minute details may mean all the difference between life and death, as unexpected autoextubation or aspiration of the tube into the trachea (impossible with the normal tube) can easily be fatal accidents. After a special tube thus carefully made has been worn a month or six weeks it usually must be changed and cleaned or the accumulation on it may cause it to clog suddenly, or produce ulceration, or a septic bronchitis, or pneumonia. Another tube should be at hand of one sixty-fourth or one thirty-second of an inch larger diameter through the neck and retaining swell than the last. Some slight improvement in the stenosis can generally thus be detected in the resistance offered to intubation if it is not clinically apparent. The judgment of the operator must determine the amount of pressure which can be borne without sloughing. Fortunately the larynx seems capable or bearing much more traumatism in these chronic conditions than one would think possible, and if some little ulceration occurs as it often must, the epithelium will in course of time cover the raw area unless the pressure is extreme. Furthermore this ulcerative process, if not too extensive, may actually be the way in which cure takes place in these cases of simple hypertrophic laryngitis. Scattered areas of fibrous cicatricial tissue may in contracting against the cartilages do away with the preceding chronic exudative and productive inflammation. A most important question in these

and to a less extent in cases having another etiology lies in the use of metal or vulcanized rubber tubes. The metal instrument is usually made of bronze or some less expensive compound heavily gold plated. At any rate it is composed of two different metals, the exposed part being gold. In two or three of the cases of hypertrophic laryngitis described above absolutely no progress could be made after weeks or months of treatment with hard rubber tubes so constructed as to make all the pressure the larynx seemed able to bear. Then after the substitution for the special rubber tube of a similar or slightly smaller compound metal tube, either a cure or very marked improvement was found at the next change or within four or five weeks. I am unable to explain this fact except on the theory of some electrolytic action. The metal instrument is far heavier than the rubber and the pressure may have something to do with its advantages, but I doubt it, as I know I have made more pressure with a large rubber than with any metal tube and there must be some other reason for the difference. The claims for electrolysis are generally regarded with suspicion, but these metal tubes, after being worn a month or two, become heavily coated wherever they press most upon the larynx, that is chiefly around the head and neck, with a stony, calcareous deposit, and under and around this the gold plating entirely disappears. There is, of course, no acid or alkali in the body which can dissolve gold, and it does not chip off but apparently dissolves. The external calcareous deposit, of course roughens the surface, and if the tube is worn for a long time may reach such proportions as to prove a very serious obstacle in extraction. I once met with such a case in the practice of a colleague in which the tube, having been in place for several months, required for removal general anæsthesia and a strong pair of forceps to permit a firm grasp of the head of the tube. The metal tube will, however, if so constructed as to exert moderate pressure upon all the thickened tissue of an hypertrophic laryngitis, sooner or later produce a cure. But the patient who wears one must be constantly watched, as it has a tendency to clog and thus asphyxiated one of my cases and very nearly did the same with another (Case XV.), and naturally after those experiences I have always been afraid of these tubes and only resort to them when the special hard-rubber instrument after thorough trial in increasing sizes proves a failure.

Autoextubation is a frequent and very dangerous complication in any intubated case, but especially dangerous in hyper-

trophic laryngitis and introduces the "special," or normal "clamped," or "plugged" tubes. Some patients will cough out any tube which has not such a mechanical attachment to keep it in place, and as autoextubation is most common with hypertrophic laryngitis, I have come to believe that some degree of this condition is, if not the primary, at least the secondary cause of the difficulty. The chronically inflamed tissues are irritable and their contact with the tube excites coughing accompanied by retching or involuntary expulsive contraction of the pharynx. The combination of an attempt to cough and vomit at the same time seems the surest way for the patient to get rid of the tube, and life generally depends on its immediate reinsertion or a hasty tracheotomy. The latter operation probably, by giving rest to the larynx, seems occasionally to produce a cure, but ordinarily it only adds a cicatrix and a possible tracheal deformity to an already bad-enough condition. If there were no other way to escape the dangers of autoextubation it might be justifiable, but hypertrophic laryngitis used to be one of the most frequent obstacles to decannulization and it seems hardly proper to inflict a tracheal fistula for its cure. Still, the tube must be kept in the larynx and for this purpose I offer the "clamped" or "plugged" instrument. If autoextubation is at all frequent the trachea is carefully opened in the median line through the second and third rings and the intubation tube while in position marked to correspond with the incision, or if a tracheal fistula has already been made the tube is inserted and marked at a point coinciding with the fistula. During the few succeeding days required to make the instrument the cannula will have to be worn, but this inflicts the least possible damage. Some explanation is required as to the comparative advantages of a "clamped" or "plugged" tube and as to the use of such an instrument in general. It is assumed that the trachea is opened carefully in the median line well below the cricoid, and only enough to comfortably admit the cannula and subsequently the retaining attachment for the tube. Deviations from these rules as indicated previously promote cicatrices and tracheal deformities. If the fistula is already existent when the patient comes for treatment, as is frequently the case, there is, of course, nothing to be gained and much may be lost by enlarging or altering the position of the fistula, and it is then only a question of a normal or "clamped" intubation tube. The self-retaining instrument is far preferable to a tracheal cannula, to which it is the only alternative in hypertrophic laryngitis complicated by

frequent and dangerous autoextubation if only because it places the patient in a practically normal condition with the single exception of a whispering voice. For less serious cases of stenosis, especially those which experience has demonstrated can breathe for an hour or two without mechanical assistance, the self-retaining tube may be thought to possess doubtful advantages. But in these there are many factors to be considered. To be sure at least one of the patients in this series could not swallow while wearing the instrument, but that experience is very exceptional, and in adults, especially "nervous" subjects, equally common with the normal tube. That the clamp or plug may act like the cannula in promoting or maintaining a tracheal deformity or favor a necrosis of cartilage has always been my fear, and hence I never use it except in cases of necessity; but experience seems to show that no ill effects are to be anticipated, and hence I have no hesitation in recommending it whenever there is the least anxiety as to autoextubation or subsequent adductor spasm. In my earlier cases, owing to unwarranted suspicion of the self-retaining tube, I believe I have taken great and improper risks in using the "special" tubes, although they are less unsightly and more ideal in effect. The perpetuation of the fistula by the "plug" also offers the distinct advantage of a sense of security in adults who are relieved of the dread of autoextubation, and in case of clogging of the tube can remove the plug and cleanse the interior of the instrument by simply poking a feather through the opening. Indeed I was first forced to employ this kind of tube in Case XV.; the normal (metal) tube previously used became obstructed one day quite unexpectedly and so nearly produced asphyxia that a substitute arrangement seemed imperative. This woman thereafter cleansed her tube in the way suggested every morning and never again had difficulty.

After removal of any tube, particularly a large or special one which has been worn a long time, there is great danger of sudden adductor spasm and strangulation and an opening in the trachea already present is a great safeguard. The spasm, however, usually occurs some days or hours after the removal of the tube (and plug), and the "fistula," when not kept patent artificially, is apt to nearly or quite close before this dangerous incident. The usual curved cannula, which could be employed in the interim, is manifestly improper, as it drags the lower segment of trachea forward and pushes the upper back, and therefore I recommend a perfectly straight cannula so fitted to its shield that it can be

pushed in or pulled out and thus stay with its inner extremity protruding into the lumen of the trachea. The diameter of the straight cannula should not be over one-eighth or one-quarter of an inch to avoid undue separation of the tracheal rings and approximation of their unsupported posterior extremities, and at the same time it must be large enough to permit, if need be, the insertion of a tracheal dilator through the fistula it maintains. This straight cannula only need be worn for the first two weeks after removal of the intubation tube. Subsequently to this period I have never met with an adductor spasm, and any other obstruction occurs slowly and can be met by reintubation.

The plugged tube cannot be made in small sizes, as either the plug or the tube or both must be constructed of vulcanized rubber, and there is a limit to the strength of this material. The plug and tube cannot both be made of metal, as the calcareous deposit which always takes place on the surface of a metal tube is very hard and firmly attached and might easily prevent the removal of the plug if the latter were likewise made of metal. The workmanship must also be of the best and very exact, because the least irregularity in correspondence between the lumen of the plug when screwed in place and that of the tube will cause rapid clogging. Possibly a metal tube with a hard-rubber plug is the best combination for cheapness, strength and durability, and as the retaining rod and cap are also of metal it offers possibilities for attempts at electrolysis for those who believe in such treatment. The "clamped" tube is the only one I have found capable of construction in the small sizes for children under ten years, and it is very useful. The tube has to be grooved on its outer surface to smoothly and exactly receive the clamp, as any irregularities or roughness will produce ulceration. The tube must be of rubber and the clamp preferably of silver, as a metal tube and a metal clamp would speedily become inseparable on account of the calcareous deposit previously mentioned. Also a clamp fashioned like an obstetric forceps is much better than one having the blades pinned together as in a pair of ice tongs. The disarticulated blades, though somewhat difficult to insert in a squirming, frightened child, will enter a very small hole and also there is no pin to break and drop into the trachea. The "cap" of metal then screws down snugly on the tube over the shanks of the clamp and should leave no rough or irregular interval to produce irritation.

The treatment of the adductor spasm, which is particularly to be dreaded after using large or "special" tubes for the cure of an

hypertrophic laryngitis, has been partly outlined in the advice given as to maintaining the patency of a tracheal fistula. It is dangerous and must always be watched for after any prolonged intubation; as it is apparently due to an atrophy of disuse in the adductors a small tube which permits them to functionate and at the same time maintain respiration is a logical procedure. After determining, if necessary, with the aid of anæsthesia that the hypertrophic condition has subsided a normal tube of the size proper for the age and development of the child is inserted and left for about one or two weeks. During this period and for a week longer assistance must be constantly near at hand in case of autoextubation. If, however, a clamped tube has been used and the small straight cannula retained in the fistula the danger is very slight. The nurse or any attendant, should difficulty occur, can readily push the straight cannula or a curved one for that matter into the trachea. The straight cannula is apt to slip outside the cartilaginous rings, especially when worn with an intubation tube. A curved cannula, by the way, inserted alongside the tube or into a fenestrum in its anterior wall, I have found to be an utter failure.

A *cicatricial* stenosis, particularly if a hasty tracheotomy has at some earlier stage been performed, is often complicated by a contracted trachea with a spur opposite the fistula; and if there has been a tracheotomy for the autoextubation following laryngeal diphtheria, there is in addition probably more or less hypertrophic laryngitis. But even when there is a history or probability of considerable loss of cartilage (see Cases VII., IX. and XV.) and contraction of the larynx until the lumen seems practically obliterated (Case XV.), experience has shown me that the prognosis is still good. In the milder cases without a tracheal fistula insertion of a normal hard-rubber intubation tube for from one to six months will invariably effect a cure. Cocaine is a sufficient anæsthetic, and if the resistance to a tube of the proper size seems too great a smaller one can be employed for a few days and then a larger substituted. In the tighter and more deforming strictures general anæsthesia is required to permit the necessary preliminary dilatation. The bad strictures always have had a tracheotomy at some earlier period or need it urgently when first seen, and the fistula serves admirably for the introduction of urethral sounds which are excellently adapted for the purpose. Beginning then with the smallest size, which will pass through the contracted area, others are passed in rapid succession

until the intubation tube can be inserted. Some judgment must, of course, be used to avoid the possibly fatal accident of making a laceration which may extend into the pharynx or œsophagus, but though I have done the operation many times and used very considerable force I have never encountered this or any ill results. Nevertheless the possibility of its happening must not be overlooked. Occasionally, as in Case XV., the stricture may be so tight or tortuous that its lumen may have to be opened by a careful dissection like a strictured urethra, but as long as any traces of mucous membrane are left and a tube can be passed from the pharynx into the trachea over or through the remains of the epithelium, there can be good hopes of a cure. Even very extensive loss of cartilage, as in Case IX., may be repaired, although Dr. O'Dwyer* cites one instance in which as the result of diphtheria the whole cricoid had sloughed out and this he considered irremediable except by tracheotomy. I should not, however, be inclined to condemn such a case without a trial.

In addition to the stricture, which is generally worse above the fistula, there will always be one at and just below this point, especially if the cannula has been worn long, and this also must be dilated like the one above. Then when inserting the tube the spur opposite such an old fistula in the posterior tracheal wall will be a serious obstacle to pass unless a small right-angled blunt retractor or hook, grooved on its convexity to receive and guide the entering tube downward and backward, is placed in the lower angle of the fistula, dragging the inferior tracheal segment forward. In adults I have several times had to exert all the force possible in two fingers pushing down from above on the head of the tube while counterpressure was made externally on the larynx with the other hand to pass this point with its spur.

The comparative advantages of using a normal, a special, or a "clamped" or "plugged" tube in these cases have already been discussed. As a rule a combination of a plugged with the principle of a special tube is the best. The plug is a safeguard against clogging and autoextubation and maintains the fistula as a subsequent escape from the difficulty of adductor spasm; the feature which for brevity I have called "special" in the instrument means an enlargement of some portion of the diameter over the corresponding diameter of the normal tube to exert a dilating pressure upon some particular part of the larynx or trachea. The normal

* American Surgery, May, 1900.

tube is long, as it was designed by Dr. O'Dwyer to pass as deeply as possible into the trachea beyond any diphtheritic process. Its lower end is constricted from side to side to pass harmlessly through the glottis, the retaining swell is as large as will pass easily through the ring of the average cricoid, and from this point upward the tube is again flattened from side to side to fit the larynx. The "special" tube must always have the retaining swell, and this can be larger than the normal, as a diphtheritic swelling within the cricoid and consequent sloughing from traumatism at this point need not be considered; but at its neck or its lower end its diameter must be increased to stretch the stricture to be combated up to the largest possible normal lumen of the air passages. No exact measurement for this can be given, and they can only be approximately estimated and by a trial of different tubes. If, for example, the stricture is in the trachea at the lower end of the cannula (rare, though I have seen one case at the Willard Parker Hospital), the tube from the retaining swell down must be of the same diameter throughout and long enough to pass the contracted area. Again, if there is an hypertrophic laryngitis complicating a cicatrix the tube must have the diameter of its neck increased, although (unless it is a clamped or plugged tube) the transverse diameter of the retaining swell must always exceed that of the neck by three thirty-seconds or preferably four-thirty-seconds of an inch.

A rough estimation of normal measurements which I have found useful is obtained by comparison with the patient's index finger, which is somewhere near the calibre of his trachea below the cricoid. The length of this finger is also about the distance from the suprasternal notch to the tracheal bifurcation.

The prognosis of a cicatricial stricture is difficult to give. Some cases seem to recover permanently after a few weeks or months of continued dilatation; others, especially those in which the rules for a proper tracheotomy have not been or could not be carried out, will relapse from time to time and prove very discouraging.

In a few instances I have attempted to dissect out all cicatricial tissue and, by accurate suture of the trachea and overlying soft parts over a well-fitting tube, to obtain primary union and so do away with these annoying and dangerous relapses. But the results have not been encouraging, possibly because asepsis is practically impossible. In Case XI., although the wound united

throughout by first intention, the stricture was again evident within a few days after the removal of the tube. In the others infection took place and the wound broke down in whole or in part. On the other hand, persistence in the dilatation for months or years (Case VII.) has seemed finally to yield a permanent cure, and hence in general cicatricial strictures of the larynx or trachea should be continuously maintained for at least six months. An attempt may then be made to do without the tube (with proper regard to adductor spasm), and at the end of a week the larynx and trachea tested for recontraction by insertion of the tube last worn. If none is apparent the test should be repeated once every one or two weeks for a month or more. At the first sign of recontraction the tube should be inserted and worn for another six months and then again tested.

But if the stricture is once allowed to contract seriously beware of attempting to intubate without preparations for tracheotomy. Such a recurrent stricture, especially if near a previous tracheal fistula, is apt to be very tight before it causes distress and very resistant to tubage. It is wisest to first attempt a passage with the Schroetter dilators, and if these fail do another careful tracheotomy in the old cicatrix when present and then dilate again with urethral sounds followed by intubation, as at first. Perhaps the safest rule after a cure seems obtained in these cases is intubation for a week every couple of months or oftener if required for several years or until no tendency to contraction can be detected during a considerable period. A pure hypertrophic laryngitis is, if anything, more difficult to handle than the cicatricial form of obstruction, as there is no time to think in the interval of changing the tube—generally for weeks or months the obstruction remains complete and the clean tube has to be immediately reinserted and it takes longer to cure the condition. But after it is once well I have never met with any recurrences of the stenosis. The average duration of treatment has been from about six months to a year, although one case recovered permanently within four weeks and another, complicated by numerous cicatrices to be sure, required nearly three years before natural breathing was finally obtained. This very long period was, however, largely due to my inexperience at that time.

Granulations as a cause of chronic obstruction are, I am convinced, rare, and when present are fortunately easily handled. Their removal, like any other new-growth, by cauterization, ex-

cision, or curettage is, of course, the first step and this can generally be accomplished through the mouth or tracheal fistula, although the latter may have to be enlarged to provide access. To prevent recurrence sources of irritation must be eradicated by making a new fistula or by changing the shape or length of the tube.

Granulations are most common at the base of the epiglottis around the head of the intubation tube, and Dr. O'Dwyer describes two cases in which they were finally overcome in this situation by so building up and remodelling the head of the instrument as to distribute the pressure it exerted over a larger area. In Case IX. the difficulty was satisfactorily met by using a smaller than normal tube. The principle, of course, is to remove the traumatism, or at least minimize it, and thus the details for individual instances can be readily adjusted.

A word as to feeding may be necessary. For the first few days after intubation liquids only are permissible and they should be taken in the Casselberry position with the mouth lower than the pharynx and it lower than the œsophagus. But it is surprising how quickly these patients learn to swallow a varied diet naturally and how few abnormalities they display. Even if an intubation tube should have to be worn permanently this is far preferable to the alternative of a tracheal cannula.

OBSERVATIONS UPON THE CAUSE AND TREATMENT OF PERINEAL ABSCESS AND OF PERIURETHRAL SUPPURATIONS ABOVE THE TRIANGULAR LIGAMENT.*

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The term "perineal abscess" has not an exact significance. It is used to describe any suppuration that involves the tissues of the perineum without regard to the extent, or cause. This term includes, therefore, the conditions described as periurethral abscess, urinary abscess, infiltration and extravasation of urine, suppurative cowperitis, periprostatic abscess. The clinical phenomena of these conditions are not the same, but the essential cause is the same in each, viz., microbic infection extending from the urethra, as the result of either disease or injury.

Perineal abscess may be associated with infiltration of urine; but infiltration of urine is not an essential causative factor. Perineal abscess may be associated with stricture of the urethra but it occurs quite frequently when stricture is not present.

Perineal suppuration occurs either as a circumscribed abscess limited to the perineum, and often to one side of the perineum, or as a rapidly spreading diffuse phlegmon which extends outside the limits of the perineum into the scrotum, the penis, and the abdominal wall. A circumscribed abscess becomes diffuse, under conditions which provoke and favor the spread of infection in the tissues; this is often the result of urine leakage.

It is to the infections of the perineum which result in circumscribed abscess, that I wish to call attention in order to define their cause, and to describe a method of operative treatment which I have used for a number of years, which differs in principle and technique from that usually followed.

The opinion is held very generally that circumscribed abscess of the perineum is not in many cases of urethral origin. It is

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claimed that it can be efficiently treated by simple incision and drainage, without opening the urethra except in cases in which there are distinct signs of urinary infiltration. This view of perineal abscess and its treatment is erroneous; but because it is so generally accepted, it is necessary to discuss the questions which underlie it.

Cases of circumscribed perineal suppuration may be divided into two groups, viz., 1. Those in which abscess occurs associated with pronounced urinary obstruction, caused by urethral stricture. 2. Those in which there is no urethral stricture. This is not simply a clinical distinction, but is in accord with the pathology of these infections. It will appear later, however, that the line between these two varieties cannot be too sharply drawn, and that there are many phenomena common to both.

Perineal abscess, when it is treated simply by incision and drainage without opening the urethra, is followed very often by a recurrence of the abscess at the original seat of the infection. Of this fact I became convinced many years ago. The cause of the recurrence I was led at first to believe was urinary infiltration; to prevent this I began to treat these cases by opening the urethra in the perineum, and draining the bladder for a few days, in addition to evacuating the pus from the abscess. In many of the cases treated, it was impossible to discover at the operation any communication between the urethra and the abscess, nor did the abscess in these cases contain any urine. In cases in which there was communication between the abscess and the urethra, the signs of infiltration of urine often were slight.

As in some of the cases in which the membranous urethra was opened, but in which there was no urinary infiltration, the abscess recurred, I began an anatomical investigation of all cases of perineal suppuration admitted into my service at Bellevue Hospital, in order to find out, if possible, the exact source of the infection, and the cause of the recurrence. I discovered that in a large number of cases of abscess of the perineum, in which the pus seemed to be altogether below the triangular ligament, and to be limited to the perineum, there were also foci of suppuration situated above the triangular ligament. These latter were found in the prostate, and alongside of the membranous urethra posteriorly. They seemed to have originated in the glands of the prostate, the glands of Littré, or the glands of Cowper.

These collections of pus did not always communicate with the perineal abscess, or at least there was apparently no communication. They were discovered only after the urethra had been opened and the posterior urethra explored by the finger. In other cases the suppuration in the perineum was unmistakably due to an extension of the infection from an abscess which began above the triangular ligament. In some of these cases the abscess had opened spontaneously into the urethra before the operation, and the pus was draining imperfectly into the canal. Unless the urethra had been opened, the presence of these abscesses would not have been suspected or discovered at the time of operation.

The recurrence of a perineal abscess in cases of urethral obstruction from stricture, might be explained by a recontraction of the stricture after operation, and subsequent urinary infiltration above the obstruction; but in a great number of cases in which the abscess recurred after an operation by simple incision, I could find no stricture, and no urinary infiltration, nor any direct anatomical communication between the abscess and the urethra. At that time it was not clear to me how pathogenic micro-organisms could invade the perineum and produce suppuration, when the urethra was apparently uninjured; nor could I explain cases of rapidly spreading diffuse phlegmon, seemingly the result of urinary extravasation, in which no opening in the urethral wall was discoverable.

I was engaged in the study of these questions, when in 1892 Delbet published his observations upon the dilatability and distensibility of the urethra, and his theory of urinary infection of the tissues about the urethra.

By a series of carefully conducted experiments upon fresh post-mortem subjects, Delbet claimed that every urethra has a physiological capacity for distention. If this is exceeded, leakage of urine into the tissues occurs through minute fissures in the mucous membrane. A comparatively feeble pressure may effect this, and he found that the most common situation of these fissures was in the perineal urethra. He explained the occurrence of periurethral abscess and perineal abscess by a microbic infection and urine leakage through these very minute fissures in the urethral mucous membrane.

This theory might explain the cases in which the suppuration is below the triangular ligament; especially those cases of abscess which occur in the course of acute inflammations of the urethra treated by forced injections or irrigations, and those

which occur from narrow urethral stricture. In the latter the fissures are caused by the pressure of the urine above the stricture where the urethra is dilated and thinned. It does not explain, however, the occurrence of perineal abscess when there is no urethral stricture, nor those cases of abscess occurring in acute gonorrhea, in which no local treatment has been used in the urethra.

In confirming the results of Delbet, I made a number of dissections of pathological specimens of perineal suppurations, and was convinced by these that in some cases at least the perineal suppuration was an extension of an infection from the urethral glands above the triangular ligament. It was not, however, until 1903 that the truth of this view was demonstrated fully by the investigations of Motz and Bartrina.

These observers undertook to investigate the cases of cowperitis preserved in the Museé Necker; and they were led to extend their study to all cases of perineal suppuration in which the preservation of the specimens made it possible to form a conclusion as to the origin of the infection.

These authors call attention to the situation of the glandular appendages of the urethra; and more especially to the great number of the glands of Littré in the membranous portion, and to the inconstant masses of glands resembling in structure the glands of Cowper situated in the bulb of the corpus spongiosum—"the glands of the bulb." They conclude that perineal abscess when not caused by mechanical tears or rupture of the urethra, is the result of an infection which begins in these glands, or in the glands of Cowper or in the prostate, and extends into the perineal tissues. But they state that in the specimens examined the infection was not confined to the glands in one situation, and that the primary source of the perineal abscess could not always be determined definitely. For example, a perineal abscess may begin almost simultaneously as either a cowperitis, a littritis, a bulbitis, or a prostatitis—and clinically it is impossible often to distinguish between a littritis and a cowperitis.*

* In eighteen specimens of stricture complicated by perineal suppuration examined, in which the source of infection could be definitely made out: In three specimens it was due to a cowperitis, a bulbitis, and a littritis; in two to cowperitis and bulbitis; in five to cowperitis and littritis; in one to cowperitis and periprostatitis. In the eighteen specimens examined there were: Four with urinary abscess; six with simple perineal suppuration; eight with infiltration of urine; but in three of these eight cases there was no opening into the urethra. Of these eight cases: In five there was a cowperitis; in one a bulbitis; in one a littritis; in one a periprostatitis.

A perineal abscess may be associated with infiltration of urine or there may be no urine leakage. When the infection from the glands extends alongside of the urethra, the abscess may open spontaneously through the mucous membrane, and urine escape into its cavity. When this occurs a simple circumscribed perineal abscess, suddenly becomes diffuse. The escape of foul and infected urine, especially of urine which undergoes rapid ammoniacal fermentation, into a simple perineal abscess, increases the virulence of the infection, and causes the very grave symptoms which are observed in the course of diffuse perineal phlegmon.

But an acute diffuse perineal phlegmon may occur with all the signs of rapid urinary extravasation, suppuration, and rapidly spreading gangrene, without any opening into the urethra, and without urinary infiltration. In this type of cases the fluid found in the tissues is not urine, but a septic edema. The intensity and extent of the phlegmon does not depend upon urine leakage, but upon the presence of a specific pathogenic micro-organism.

In certain cases of circumscribed abscess, the evolution of the infection is slow, and the urine infiltration is slight. In these the tissues of the perineum are gradually transformed into hard masses of inflammatory tissue, called "urinary tumors." These often open upon the surface of the skin of the perineum and scrotum, and fistulous tracts are formed from which urine escapes. This condition usually occurs in cases of narrow stricture which have been neglected.

The observations of Motz and Bartrina and those of Delbet, I have confirmed in many particulars. They correspond with my clinical experience, and are in accord with the general principles of the operative treatment which I have employed with increasing success for more than ten years, in all cases of perineal suppuration. I call attention to this operation, since I can recommend it now not only as justified by the clinical results obtained, but as being in accord with our present anatomical and pathological knowledge of perineal suppurations.

The view that perineal abscess in cases of narrow stricture is due to ulceration of the urethra above the point of stricture, cannot be accepted. It is only necessary to study these cases post-mortem to find that the ulcerations are usually very small and superficial, and confined to the mucous membrane; they rest upon an indurated base, the result of a protective inflammation, which

is impervious to liquids. An ulceration sufficiently rapid in its evolution to permit the escape of urine from the urethra into the surrounding tissues is the result of periurethral suppuration or of mechanical injury.

The source of the infection in a perineal abscess I believe is always from the urethra. The infection may occur in three ways: (1) Through the glands connected with the bulbous or membranous portion, *i. e.*, the glands of the bulb, the glands of Cowper, or Littré's glands, or the prostate; and from these extends into the perineal tissues; (2) it may occur through small fissures in the mucous membrane of the urethra (Delbet); (3) it may be the result of a more or less extensive mechanical rupture of the urethra from external or internal violence. Infiltration of urine occurs and plays a prominent part in the infection of some cases, but often it is absent. It is not an essential causative factor in all cases. Stricture of the urethra may or may not be present.

Delbet's views as to the ease with which the mucous membrane of the urethra may become fissured by slight overdistention, makes it evident that forced irrigations and injections favor periurethral infection. This has been my experience clinically. Irrigation of the entire urethra from the external meatus without using a catheter is, I believe, bad, both in principle and practice. The deep urethra should not be irrigated except through a catheter. Injections should never be used forcibly, and in quantity should not exceed the normal capacity of the urethra. There are some cases of perineal abscess which occur during the course of an urethral inflammation, in which the immediate exciting cause of the suppuration is not apparent. Abscess occurring in acute urethritis in which no local treatment has been used, is an example of these.

Periurethral suppuration beginning in the urethral glands may continue for a long time before the infection extends into the perineum; this is especially true in cases of narrow urethral stricture and in cases in which there has been repeated attacks of urethritis.

An early diagnosis of periurethral suppuration is of great importance. In all cases in which perineal infection may occur, the prostate, and the membranous urethra, should be examined by rectal touch, in order to detect the first signs of suppuration; and the perineum should be carefully examined by rectal and external touch combined.

When there is periurethral suppuration above the triangular ligament, originating either in Cowper's or Littré's glands, there is a fullness and often fluctuation found in front of the apex of the prostate, either on one or on both sides of the membranous urethra. There is also an edematous condition of the tissues of the anterior wall of the rectum. The edema sometimes extends up over the posterior surface of the prostate. When the prostate is the seat of an abscess, there is an increase in its size and changes in its outline which can be detected by rectal touch even in cases in which the abscess has opened spontaneously into the urethra, or in those in which fluctuation is absent.

When a perineal abscess originates in Littré's or Cowper's glands and subsequently invades the perineum, the course which the pus takes is along the wall of the membranous urethra, and it invades the perineum close to the opening in the triangular ligament. The communication between the suppuration above the triangular ligament, and the perineal abscess, is usually by a sinus which is found above and on one side of the bulb and close to the opening through which the urethra passes.

When a littritis and a bulbitis occur simultaneously, the suppurating focus above the triangular ligament may open spontaneously into the urethra and not communicate with the perineal abscess; the latter being due to extension of infection from a bulbitis. Suppuration about the membranous portion of the urethra, arising from a littritis, or a cowperitis cannot be efficiently drained without opening the membranous portion of the urethra.

When suppuration begins above the triangular ligament, it may extend into the ischiorectal fossa, as well as into the perineum. This occurs in one or two ways: The pus burrows either beneath the posterior border of the triangular ligament, a little to one side of the middle line and passes directly into the ischiorectal space; or it invades the perineum first and later on passes the limits of the deep layer of the superficial fascia, as the result of necrosis of this membrane.

Ischiorectal abscess associated with perineal abscess occurs with or without urinary infiltration; its occurrence seems to be frequently associated with the latter. I have met, however, with rapidly spreading infections from Littré's glands and Cowper's glands, which destroyed the deep layer of the superficial fascia, and extended into the ischiorectal fossa, without finding any

communication between the perineal abscess and the urethra. In one of these, Case VII., this layer of the perineal fascia was found perforated by irregular necrotic areas, the tissues were gangrenous, but there was no evidence of urinary infiltration.

Perineal abscess is often associated with suppurating foci in the prostate, and these are found only after opening the urethra. The abscess may have opened spontaneously before operation into the urethra. The exact relation between prostatic abscess and perineal abscess was for a long time difficult to explain; recent observation has convinced me that the latter is caused usually by the extension of infection from Littré's or Cowper's glands, or the glands of the bulb, coexisting with the prostatic suppuration.

It is only within the last three years that I have discovered that the cases of perineal abscess which formerly I thought were caused by prostatic or periprostatic suppuration, are due to infection extending from the urethral glands, which are involved coincidently with the prostatic abscess.

The symptoms and physical signs of perineal abscess can be gathered from the histories of the cases appended to this paper, and it is unnecessary to consider them at length. I wish, however, to call attention to the necessity of making a thorough examination for the beginning of periurethral suppuration, even before the infection has invaded the perineum. In cases of difficult and painful urination, when there is deep seated pain, or tenderness in the perineum; or when there is a perineal swelling, an examination by rectal and external palpation combined should be made to detect suppuration. It is not necessary to wait until an abscess is fully formed in the perineum, and fluctuation is present to decide the diagnosis. As soon as periurethral infection occurs, an operation is indicated, to prevent further extension, and to hasten the patient's recovery.

There are many at the present time who believe that infection of the urethral glands and of the prostate can be treated successfully without operation. It is a waste of time to employ rectal irrigation or massage or any of the numerous local measures so often employed in the treatment of these conditions. The cases which seem to get relief from these measures usually later on develop abscess; or, if they are relieved, it is by the spontaneous opening of the abscess into the urethra; the opening in many cases is insufficient to drain the abscess, and so these cases re-

main with an incurable urethral discharge, and in addition are exposed to the danger of recurrence of the abscess.

The Operative Treatment of Perineal Abscess—This should be in accord with the anatomy and pathology of these suppurations. The treatment of these cases generally is defective, because the necessity of opening the membranous urethra and of draining the pus foci which exist above the triangular ligament is not understood or is disregarded.

It does not seem to be understood that perineal abscess is either directly the result of periurethral suppuration above the triangular ligament, or is in nearly all cases associated with suppuration in this situation as the result of coincident infection of the urethral or prostatic glands. It is necessary therefore to open and drain the perineal abscess, and also to open the membranous urethra, and to drain the foci of suppuration above the triangular ligament.

A perineal abscess is opened by a free median incision extending from the scrotum to three-fourths of an inch in front of the anus. The deep layer of the superficial fascia is divided for the entire extent of the incision, and the pus of the abscess is evacuated. The abscess cavity is cleansed of all necrotic tissue, and any sinuses or pockets are opened. The membranous urethra is opened upon a staff with a wide median groove; this incision should divide the floor of the canal and the lower border of the trianguar ligament, and should extend from the bulb of the urethra to the apex of the prostate. The forefinger of one hand is passed into the urethra through the perineal incision as the staff is withdrawn. A careful and systematic exploration is then made for any foci of suppuration: and it is sometimes useful to make counter-pressure with the finger of the other hand upon the anterior wall of the rectum, during the examination.

When there is suppuration in or about Cowper's glands or the glands of Littré, a soft, sometimes fluctuating, spot is felt at the side of and below the membranous urethra. If these pus foci have opened spontaneously into the urethra the opening or openings can be felt as round or oval holes on the side, and sometimes near the roof of the canal. If the mucous membrane of the membranous urethra is still intact over the abscess at the time of operation, it is to be gently torn through by the fingernail. If the abscess has opened spontaneously before operation, the openings in the urethra are united by tearing through the bands of

diseased mucous membrane which separate them. The tip of the finger is passed into the abscess cavity, which can be explored. This should be made level with the floor of the membranous urethra by gently tearing with the fingernail the mucous membrane toward the perineal wound; the pus can then drain into the incision made in the urethral floor. Abscess in the prostate is similarly treated when present. The bladder and wound are washed out, and the former is drained by a large catheter, No. 28 to 30 F. The perineal abscess cavity should be drained with a few strips of iodoform gauze.

The drainage tube is retained for two, sometimes for three days, and after its removal the case is treated as any perineal section. A steel sound as large as the external meatus will admit without pain is passed at the end of one week and once a week afterwards until the perineal wound has closed. In cases of narrow stricture, the urethra at the time of operation, is rapidly dilated by tunneled sounds passed over a whalebone guide until it can admit an Otis urethrotome; then after opening the membranous urethra the stricture is cut by internal urethrotomy upon the roof of the urethra. In cases of diffuse perineal phlegmon, the same method of treatment is followed, but in addition, free incisions through infected areas of the skin and cellular tissues should be made wherever there are signs of "infiltration of urine."

In cases of traumatic rupture of the urethra from external violence, an early operation is imperative, even before there is any perineal infiltration; but I am opposed to the plan of attempting to unite by suture the divided ends of the urethra.

Objections which can be raised to the method of treatment just described on theoretical grounds, are answered by the results which have followed its use in my service at Bellevue Hospital. An abstract is appended of the histories of a few cases which have been taken from a very large number treated in this service, to illustrate the points which I have endeavored to make clear in this paper. The success of treatment depends upon the surgeon's knowledge of the anatomy of these conditions, which cannot be learned from a written description, but must be acquired by personal clinical experience and observation.

Case I.—*Relapsing Perineal Abscess Due to Cowperitis; Slow Urinary Infiltration; Stricture; Operation; Recovery*—H. G., age 26, United States. Admitted November 12, 1901. Three years before admission he had a

gonorrhea; he was treated by injections. An abscess formed in the perineum; this was opened in a hospital at Sheffield, England. The wound healed in three weeks. Two years later he contracted a second gonorrhea, which was treated by injections; in the seventh week of the disease a swelling appeared at the site of the old abscess in the perineum; this subsided without operation. Three weeks before his admission he contracted a gonorrhea for the third time. Two weeks later a swelling appeared in the perineum, which became very painful, and two days before admission to the hospital it broke through the skin and discharged pus.

He had noticed that for some time his stream was diminished in size, and he had some frequency in urination. There was present a swelling in the perineum on the left side of the urethra, which was quite hard, but there was deep fluctuation; leading into this swelling was a sinus, which did not discharge urine.

Operation on November 13. An abscess was found beneath the deep layer of the superficial fascia on the left side. A sinus was found leading into the urethra at the bulbomembranous junction. The membranous urethra was opened and a cavity was found on its left side, due to pericowperitis; this was opened into the urethra and the bladder was drained. A stricture at the bulb was cut internally to No. 30F. He was discharged cured on February 22, 1902.

Case II.—*Abscess of Perineum, Due to Cowperitis and Littritis; Two Previous Attacks, one of Which Resolved; in the Other the Abscess Opened Spontaneously in the Perineum; no Stricture; no Urinary Infiltration; Operation; Cure*—E. R., age 31, United States. Admitted December 12, 1901. He had gonorrhea thirteen years before admission and again ten years later; the latter was rebellious to treatment, and at the end of the fifth month a swelling appeared in the perineum, which was painful; this subsided after a few days without operation. A year later he still had a urethral discharge, and a swelling reappeared in the perineum; this became hot and painful, and finally ruptured externally, discharging a large amount of pus, and he thinks that for a week some urine passed through the wound, but the sinus healed spontaneously.

One week before admission he contracted gonorrhea; the urethral discharge was profuse, and three days before admission a swelling appeared in the perineum. In the perineum there was a swelling about the size of a pigeon's egg, beneath the deep layer of the superficial fascia. Rectal examination showed the prostate normal in size; there was fluctuation about the membranous urethra and in the perineum. He had pain on urination; no obstruction to urination; no increased frequency.

Operation December 13. Perineal section: An abscess was found beneath the deep layer of the superficial fascia, in the lower portion of the abscess cavity; close to the bulb a sinus was found leading up to situation of Cowper's glands. The membranous urethra was opened and the abscess drained. No communication could be found between the urethra and the perineal abscess. The patient remained in the hospital for some weeks after his recovery, and was discharged February 22, 1901, cured.

Case III.—*Perineal and Ischiorectal Abscess Originating in Either Cowper's or Littré's Glands; Urethral Stricture; no Infiltration of Urine; Operation; Cure*—D. McK., age, 31, United States. Admitted April 9, 1902. He had had four attacks of urethritis. One and one-half years before admission he noticed that his stream was becoming smaller; three months later he had retention of urine and was operated upon for stricture at Hudson Street Hospital, New York, by perineal section. Sounds were passed twice a week for two months after the operation. Ten days before admission he developed a urethritis; a week later a left epididymitis. He passed urine four times a day, twice at night; he had no chill and no retention. The day before admission he noticed swelling in the perineum, with pain on walking and pain on defecation. The swelling was confined beneath the deep layer of the superficial fascia. Rectal examination showed the prostate of normal size; there was marked edema of the anterior rectal wall; on the left side of the membranous urethra there was a swelling which extended out to the left. There was fullness and sense of fluctuation in the perineum on the left side, which extended down into the left ischiorectal fossa.

Operation on April 10; Median perineal section; about one-half ounce of pus was evacuated from beneath the deep layer of the superficial fascia. The membranous urethra was opened; no communication was found between the perineal abscess and the urethra, but an abscess was found on the left side of the membranous urethra and another in the ischiorectal fossa; these were drained, and the patient was discharged, cured on April 20.

Case IV.—*Acute Perineal Abscess from External Violence; Retention of Urine; Urinary Infiltration Without Apparent Opening into Urethra; Operation; Cure*—A. W., 37, Swede. Admitted the night of October 18, 1904. He had gonorrhea seventeen years before; nine days before admission he fell astride an iron rail, striking upon his perineum. He had severe pain in perineum at the time, and the next urine which he passed was bloody. During the following night he had to urinate every two hours, and passed blood at each urination. Pain in the perineum increased; his desire to urinate became more frequent. He noticed swelling in the perineum, and on the day of admission to the hospital he had retention of urine.

Examination showed a swelling of the perineum, extending up into the scrotum; the skin was red and edematous. A soft rubber catheter was passed and the retention was relieved. The operation was delayed until the following morning, October 19. External urethrotomy. An abscess was found beneath the deep layer of the superficial fascia; it was more on the right side than on the left side of the urethra. A communication between the abscess cavity and the urethra could not be found. The membranous urethra was opened. The bladder was drained. The patient made a good recovery and was practically well a month later, but had a small fistula, which persisted for nearly two months and then healed.

Case V.—*Perineal Abscess; Bulbitis; Urinary Infiltration; Urethral Stricture; Operation; Cure*—T. B., age 23, United States. Admitted No-

vember 16, 1904. He had a gonorrhea fourteen years before; and symptoms of stricture with difficult urination for the past nine years, for which he had been treated by dilation with sounds. On November 10 he noticed pain in the perineum; this increased from day to day and was most severe during urination; he had no pain on defecation. On November 14 he noticed for the first time, a swelling in the perineum. The pain became so severe that he came to hospital for relief.

On admission he had almost complete retention of urine. There was a marked swelling beneath the deep layer of the superficial fascia with edema of the scrotum. A narrow stricture of the urethra admitting a filiform guide was found just in front of bulb.

Operation on November 17. Perineal section: Stricture was cut internally to No. 30 F. after first being rapidly dilated to No. 20 F. The perineal abscess communicated with urethra at bulb by rather large opening; source of infection probably in accessory glands of the bulb. Discharged, cured, December 17, 1904.

Case VI.—*Case of Perineal Abscess, Due to Cowperitis Associated with Prostatic Abscess; no Urinary Infiltration; Operation; Cure*—W. J. M., age 28; United States. Admitted April 5, 1905. He had a gonorrhea when twenty years of age, and again two years later. He was treated by irrigations almost continuously for a year and sounds were passed at intervals. Four days before his admission he began to have pain in perineum, increased by walking and by sitting down. He had no pain or difficulty at urination. On the next day he noticed a swelling in the perineum.

On admission he had a swelling in the perineum more marked on the left side, but which extended somewhat beyond the median line. This was tender; the skin over it was red; it was about one and one-half inches long by three-fourths inches wide.

Operation April 5. Perineal section. The patient's urethra easily admitted No. 30 F. sound. There was no stricture. Beneath the deep layer of the superficial fascia an abscess was opened containing about two ounces of pus. The membraneous urethra was opened, and on passing the finger into the urethra an abscess cavity was found on the left side, extending below and alongside of the membranous urethra. Another abscess was found in the left lobe of the prostate; this had opened into the prostatic urethra. These abscesses were drained into the urethra. The bladder was drained for twenty-four hours. The patient was discharged at his own request, on the tenth day, passing all urine through the urethra, and at the end of the third week was entirely well.

Case VII.—*Perineal and Ischiorectal Abscess; Cowperitis and Littritis; no Infiltration of Urine; Operation; Cure*—F. S., age 27. Admitted May 14, 1905. He had a gonorrhea twice, the first attack eight years before, and the second three months before admission. He was treated for the latter by injections for about five weeks. Three weeks before admission he began to have pain, and a swelling appeared in the perineum. He had no frequency or pain on urination and no difficulty in passing water. He had a moderate discharge from the urethra; the urine contained free pus and shreds. Examination showed a swelling on the left side of the perineum; this extended a little to the right of middle line.

There was swelling over the ischiorectal fossa. Rectal examination showed tenderness and fluctuation about the membranous urethra.

Operation on May 15; Perineal section. On cutting through the skin down to the deep layer of the superficial fascia, this was found to be necrotic; there was a large abscess cavity on the left side of the urethra, which had burrowed also into the ischiorectal fossa. There was no communication between the perineal abscess and the urethra. After opening the membranous urethra an abscess was found on left side which apparently began in Cowper's gland. The perineal abscess was cleaned, disinfected, and a counter-opening made to drain the ischiorectal fossa. On June 10 all urine was passed through the urethra.

Case VIII.—*Perineal Abscess Caused by Double Cowperitis and Prostatic Abscess; Probably Tuberculous; Operation*—E. Q., age 28. Admitted May 14, 1905. He denied all previous venereal disease. Three weeks before admission he contracted a gonorrhea. He was treated with injections by a physician and from the first had pain in the perineum and along the urethra; he had frequency of urination, but had never passed blood; he had pain on defecation. Six days before admission he noticed a swelling in the perineum; this was painful and was growing progressively larger. He had no difficulty in passing water. He had a profuse discharge from the urethra.

Examination showed a swelling in the perineum situated beneath the deep layer of the superficial fascia with fluctuation. Rectal examination showed the prostate enlarged on the left side; tender; the membranous urethra and the tissues surrounding it were soft and edematous. The urethra was not strictured.

Operation on May 15. Perineal section. On cutting through the deep layer of the superficial fascia an abscess cavity was opened; on exploring the deep urethra a large cavity was found on the side of the membranous urethra; this communicated with the abscess in the perineum; there was also an abscess in the prostate. The suppuration seemed to have begun in Cowper's glands and this was associated with prastatic abscess. A tube was introduced and the bladder drained for forty-eight hours. The perineum healed without complications. All urine was passed through the urethra after May 26.

Case IX.—*Mechanical Rupture of Urethra; Slow Infiltration of Urine; Circumscribed Perineal Abscess; Traumatic Stricture; Operation; Cure*—J. E., age 26. Admitted May 15, 1905. He had gonorrhea seven years before admission and a second attack three years later complicated by an epididymitis. Three months before admission he fell from a height of thirty feet, striking on the perineum and inner side of thigh. He had difficulty in passing urine, and passed bloody urine. He did not have complete retention of urine, but noticed that his stream was for some time becoming progressively smaller. One month before admission he noticed a painful swelling on the right side of the perineum; the pain was increased by walking and by sitting down and by defecation.

Examination showed a deep-seated swelling on the right side of the perineum; this was painful upon pressure. It was hard, and there was no sense of fluctuation. There was no enlargement of the prostate. A

narrow stricture, admitting only a filiform whalebone guide was found at the bulb of the urethra.

Operation on May 16. Perineal section. The stricture was cut internally to No. 30 F. with an Otis urethrotome, after rapidly dilating the urethra to No. 18 F. A small circumscribed abscess was found just above the bulb on the right side; this was opened; no communication could be found with the urethra; the infection seemed to have extended from the glands of the bulb. The bladder was drained by perineal tube. The tube was retained for forty-eight hours. The patient passed all urine through the urethra on May 27. Discharged May 31, and was entirely well on June 5.

PROSTATIC AND PERIPROSTATIC ABSCESS—A CONTRIBUTION TO THE STUDY OF THESE CONDITIONS AND THEIR TREATMENT BY OPERATION.*

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Prostatic abscess occurs frequently, but in many cases it is not recognized until the suppuration has destroyed important structures. It is customary to describe suppurations in and about the prostate and the operative treatment of these conditions in general terms, and these descriptions often are vague and unsatisfactory.

In presenting this subject it is necessary to define the exact meaning of the terms prostatic abscess and periprostatic abscess, because these are not used with the same significance by all writers.

A prostatic abscess is a localized collection of pus within the capsule of the prostate, due to an infection extending from the urethra. The abscess may be single or there may be multiple abscesses.

Periprostatic abscess is an extension of the infection from a prostatic abscess outside of the capsule. The pus in a periprostatic abscess may be confined above the triangular ligament, but it may extend into the perineum or ischiorectal region.

Prostatic and periprostatic abscess may occur as the result of tuberculous infection or as the result of malignant disease.

Abscess of the prostate occurs most frequently from the extension of an acute or of a chronic infection of the posterior urethra, especially in those who have had repeated attacks of gonorrheal urethritis. The improper use of injections or irrigations or ill-advised instrumental treatment of the urethra often is the exciting cause. Prostatic abscess may occur as a complication of urethral stricture and usually in these cases it is the result of the

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frequent or violent use of instruments. It is not an uncommon complication of prostatic enlargement and occurs especially in those who habitually use a catheter. It is frequently the cause of sudden retention of urine in these cases.

A description of the pathological anatomy of prostatic abscess presents some difficulties. Post-mortem specimens of the disease except in its later stages, rarely come under observation.

From the examination of a large number of cases at the time of operation and of a number of post-mortem specimens, I am convinced that prostatic abscess usually begins by the extension of an infection from the urethra into the gland ducts of the prostate. The infection sometimes may occur through a wound of the surface of the prostatic urethra, but infection through the gland duct is more common. The suppuration may be confined to one or more of the excretory ducts, or the infection may extend into the gland tubes and involve one or more of the prostatic lobules, and by extension it may destroy more or less of the entire gland.

Each lobule of the prostate is composed of a number of tubular glands; the excretory ducts from these empty into the urethra on each side of the verumontanum. The excretory ducts are formed by the union of smaller ducts and these latter communicate with the terminal gland tubes. The secretion from the tubules in each lobule is discharged into the urethra through one excretory duct. Infection, therefore, of a single duct should affect the tubes in only one lobule, but when there is infection of more than one duct the foci of suppuration are multiple, and these different foci frequently open into each other and form a single abscess.

Prostatic abscess is not caused always by the same infective agent—although the gonococcus seems to be the most frequent cause. The infective agent is sometimes among the least pathogenic of the pyogenic cocci, and this I think should lead us to attach much importance to the various accessory causes which render the urethra and prostate incapable of resisting even these mild pathogenic bacteria.

The non-bacterial factors are as important etiologically as are the essential agents of infection. Among these the most active in causing prostatic abscess is the modern treatment of gonorrhœa by irrigation of the urethra from the external meatus; and the too frequent and unnecessary use of instruments in the urinary passages which is so common at the present day.

There seems to be much confusion as to the different kinds of prostatic inflammation, and clinically abscess is often mistaken for acute prostatitis. When an infection from the posterior urethra extends into the prostatic ducts, and preceding the formation of abscess, there is an increase in the size of the prostate. This is caused by retention of the secretion in the lobules communicating with the infected ducts which are obstructed by the swelling of the mucous membrane of the urethra and by an accumulation of inflammatory exudate. The gland tubes are distended and impede by pressure the circulation in the veins. The œdema of the prostate is increased by anything which increases the congestion as, for example, by the frequency of urination and the straining to empty the bladder which are so characteristic of cases of acute prostatitis. This swelling of the prostate caused by retention of the secretion in the glands and by œdema may subside without the formation of an abscess. The excretory ducts discharge the secretions into the urethra; the obstruction to the venous circulation is thus relieved, and the swelling of the prostate subsides. Small abscesses may occur and open into the urethra and may thoroughly discharge their contents; but a small abscess which has opened into the urethra by an opening too small to efficiently drain it may be the source of great distress and annoyance to the patient. A persistent urethral discharge which relapses frequently is due often to this cause and there is always the danger of recurrence, or of an extension of the abscesses. Many cases of relapsing posterior urethritis, and many which are called chronic follicular prostatitis, are really cases of imperfectly drained prostatic abscess. A prostatic abscess may be confined within the capsule for a long time, sometimes for several weeks, without opening into the urethra. An abscess freely drained by a large natural opening into the urethra often may heal without operation, but abscesses of the prostate which open into the urethra spontaneously often require operation.

Le Dentu's statement* that abscesses of the prostate which open spontaneously into the urethra usually do so by openings proportionate to the size of the abscess cavity is contrary to my experience. Therefore when an abscess of the prostate opens into the urethra spontaneously an operation is required, unless the drainage is efficient.

* Des Abcès Chauds de la Prostate et du Phlegmon Periprostatique. Segond, Paris, 1880, page 53.

The cavity of the prostate abscess as felt by the finger through a perineal opening is irregular in shape and this is especially marked when several smaller abscesses unite together. The cavity is traversed by fibrous bands which are formed of the stroma of the prostate.

Infiltration of urine does not occur always into abscess cavities which open spontaneously into the urethra, even when the openings are of large size; but when infiltration of urine does occur, the intensity of the infection usually is increased, and the suppuration extends more rapidly.

Abscess of the prostate begins in that portion of the lateral lobe which lies behind and at the side of the urethra, and the infection usually extends towards the apex of the gland. When an abscess opens spontaneously into the urethra, the opening is found upon the floor or upon the side of the urethra. It is not difficult to explain why abscess of the prostate should open in this situation; the pus follows the line of least resistance and finds its way to the urethra along the fibrous bands which separate the lobules and which converge behind the urethra at the central nucleus. The mucous membrane of the prostatic portion of the urethra upon the floor and below the verumontanum is thin, and is closely attached to the prostate; in the supramontanal region of the prostatic urethra the mucous membrane is thicker. Abscess may occur in both lateral lobes simultaneously, but usually it is confined to one side of the prostate.

When an abscess of the prostate does not open spontaneously into the urethra, but extends outside of the capsule, the pus is found between the rectum and the prostate; it may extend into the perineum or ischiorectal fossa, and in some rare cases it may find its way into the prevesical space. I believe that periostatic suppuration is due always to neglect or to a failure to recognize the original seat of the disease, and that it can be prevented by an early operation. The spontaneous opening of a prostatic abscess or periprostatic abscess into the rectum is regarded by many writers as a common occurrence, but this seldom happens, except in cases in which operation has been too long delayed.

I have observed that when a prostatic abscess extends into the perineum there is also suppuration about the membraneous urethra which begins in or about Cowper's glands or the glands of Littré, and that the latter is the cause of the perineal infection.*

*This observation has been confirmed by Motz and Bartrina. Ann. des Mal. des Organ. Gen Urin., November 1, 1903, page 1624.

In some cases the perineal suppuration seems to have extended from infection of the intrabulbar glands. I have seen not a few cases of perineal abscess in which suppurating foci above the triangular ligament were detected only after the membranous urethra had been opened and the prostate explored by the finger. In many cases of ischiorectal abscess the suppuration originates in the prostate. Segond has given an interesting explanation of the course which the pus from a periprostatic abscess follows in reaching the perineum and ischiorectal fossa.†

If a prostatic abscess opens spontaneously into the urethra, and extends also outside the capsule, and opens either into the rectum, the perineum, or the ischiorectal fossa, troublesome fistulæ may result which could have been prevented by an early operation.

The symptoms of prostatic abscess and of periprostatic suppuration vary in different cases and their intensity depends upon the character and extent of the infection and the clinical conditions under which they occur. The symptoms of an abscess which occur in the course of an acute gonorrheal infection of the urethra differ from the symptoms of an abscess in an old prostatic. A small abscess situated close to the urethra causes more suffering than a large abscess which has invaded the periprostatic tis-

† Des Abcès Chauds de la Prostate et du Phlegmon Periprostatique, Segond, Paris, 1880, pages 86, 87.

"Les données de l'anatomie topographique montrent les différentes voies qui s'offrent au pus lorsqu'il veut sortir de sa loge. Une préparation fort simple permet d'acquérir à cet égard des renseignements précis. Il suffit d'ouvrir la loge rétro-prostatique par son bord supérieur et d'explorer ses parois pour reconnaître immédiatement quels peuvent en être les points faibles. Voici comment il faut procéder:

"Le bassin d'un sujet, jeune ou vieux, mais sans lésion abdominale, étant séparé du tronc, le rectum sectionné et lié a son extrémité supérieure et les organes du petit bassin soigneusement respectés, on attire la vessie en avant, de manière à mettre en évidence le fond du cul-de-sac recto-vésical. En ce point, on pratique sur le péritoine une incision transversale, longue de deux travers de doigt, puis, on introduit son index par l'incision, entre le rectum et l'aponévrose prostato-péritonéale. La laxité du tissu cellulaire est telle, que l'on croit pénétrer dans une bourse séreuse: sans le moindre effort, on arrive sur la face supérieure de l'aponévrose moyenne. Là, on éprouve un arrêt, et le doigt ne peut plus avancer. En arrière, on sent la paroi molle et dépressible du rectum; en avant, la prostate et les vésicules. Sur les côtés enfiin, on est bridé par les solides attaches rectales des aponévroses latérales de la prostate. Si maintenant, par des pressions exercées avec la pulpe de l'index, on essaye de forcer les limites de la loge, on constate ce qui suit:

"Sur les côtés, dans tout leur hauteur depuis l'aponévrose moyenne jusqu'au niveau de la base des vésicules: résistance absolue, solide, pour ainsi dire infranchissable.

"En bas et sur la ligne médiane: résistance médiocre; l'index pénètre sans trop d'effort et gagne le périnée antérieur.

"En bas et en dehors; résistance très faible; une pression légère conduit immédiatement en pleine fosse ischio-rectale.

"En avant et sur les côtés, on est barré par la jonction de l'aponévrose prostato-péritonéale et des aponévroses latérales, ou, mieux, par l'adhérence intime de ces dernières avec la glande."

sues. A prostatic abscess cannot be distinguished always by its symptoms alone from an acute prostatitis, and a positive diagnosis can be made only by direct examination of the parts. It requires considerable practice to detect the presence of pus positively in every case.

The principal symptoms of acute prostatic abscess are partial or complete retention of urine, pain, and rectal tenesmus. These symptoms occur also as the result of other diseases of the prostate and the history of each case is necessary to a diagnosis. The retention of urine may develop slowly, or it may come on suddenly depending upon the acuteness of the infection and the situation of the abscess.

At the beginning of an acute prostatic abscess there is usually a distinct chill or chilly sensation; coincident with this chill or following it the temperature rises, but usually the fever does not exceed two or three degrees above the normal. I have noticed in many cases that in twenty-four or forty-eight hours the temperature falls to normal or to nearly normal and so remains. This fact is sometimes misleading, because the majority of authors describe as one of the symptoms of prostatic abscess and periprostatic abscess a continued and irregular rise and fall of temperature; this occurs only exceptionally, and is an indication of a general septic infection.

The spontaneous opening of a prostatic abscess into the urethra is accompanied by an unusual flow of pus, the quantity depending upon the size of the abscess and the size of the opening. The pus appears at the meatus and also flows backwards into the bladder, and then appears with the last urine passed at each urination. In these cases, when the drainage is imperfect this symptom persists. When the drainage is efficient, the quantity of pus rapidly diminishes, and this is associated with a diminution in the intensity of all the other symptoms.

Abscess of the prostate is often ruptured into the urethra during the rectal examination incident to diagnosis. In some cases slight pressure upon the prostate causes a flow of pus from the meatus. An abscess frequently is ruptured into the urethra during catheterism practised for the relief of the retention of urine. When abscess of the prostate occurs as a complication of prostatic enlargement, the retention of urine becomes complete, catheterism becomes more difficult, and is painful; the frequent use of the catheter is imperatively demanded and the suffering

of the patient is great. In cases of this kind perineal prostatectomy is the operation of choice.

The symptoms of pain and retention are sometimes relieved when the pus from the prostatic abscess extends outside the capsule. The suppuration does not extend often into the perineum or into the ischiorectal fossa unless the abscess in the prostate is associated with abscess in Cowper's or Littré's glands or the intrabulbar glands of the urethra, and I believe that in nearly all cases the perineal suppuration begins in these glands.

An examination by rectal touch is necessary to a positive diagnosis of prostatic abscess, and this always should be combined with counter-pressure first over the pubes and then upon the perineum. In the earlier stages of abscess it is impossible always to distinguish between the congestion and œdema of acute prostatitis and that of abscess, but I believe that acute prostatitis without suppuration is rare when the symptoms have existed for more than a few days and when there is complete or partial retention of urine. In an abscess confined to one side of the prostate, the affected lobe is usually more swollen than the other. Fluctuation or a point of softening is present in many cases, but, unless the abscess is large and the pus near the posterior surface of the gland distinct fluctuation cannot always be detected by rectal touch. If one waits for fluctuation before operating, much valuable time will be lost and important structures destroyed. I have found extensive abscess of the prostate in cases in which by rectal touch I had failed to find fluctuation.

The outline of the prostate in cases of abscess is sometimes obscured by œdema of the tissues in the anterior wall of the rectum. This is true especially in cases of periprostatic suppuration and in cases of prostatic abscess associated with suppuration about the membranous urethra.

In most cases of prostatic abscess I have found that the urine contains a larger percentage of albumen than can be accounted for by the pus. This symptom is one to which I attach considerable importance in doubtful cases. The albumen seems to be the result of an acute inflammatory œdema, the serum escaping into the urethra.

Abscess of the prostate should be recognized and operation performed before the pus has extended beyond the capsule, and when possible before the abscess has opened into the urethra. I am convinced that I have shortened the duration of the disease

by operating before the signs of fluctuation could be detected by rectal examination, and in all of these cases I never have failed to find one in which there was not suppuration.

An operation is indicated at the earliest possible moment unless the abscess has opened into the urethra, but in many cases in which this has occurred, I believe an operation will hasten the recovery. An abscess which has opened into the urethra by a small opening requires operation quite as much as one which has not opened into the urethra. A periprostatic abscess always requires operation.

In treating prostatic and periprostatic abscess by operation, all suppuration foci should be freely opened and thoroughly drained; this should be done by the most direct route, with the least possible injury to surrounding structures. To this general principle I wish to add that in every case the membranous urethra should be opened and the prostatic urethra should be explored through a median perineal incision.

The principal operations which have been used in the treatment of prostatic abscess are:

1. Incision and drainage through the anterior wall of the rectum.

2. Mechanical opening of the abscess by means of a sound passed into the urethra.

3. Incision through the capsule and sheath after exposing the posterior surface of the prostate by a curved prerectal incision.

4. Drainage through a median perineal section.

It would be unnecessary at the present time to speak of opening a prostatic abscess by incision through the anterior wall of the rectum; or of opening an abscess of the prostate by an instrument passed into the urethra, except that these methods are still recommended in some modern text-books which have the imprimatur of recognized authority, it is necessary therefore to call attention to these operations even if it is only to condemn their use for reasons which should be apparent to any intelligent modern surgeon. The operation of opening an abscess through the sheath and capsule after exposing the posterior surface of the prostate by a prerectal incision is that followed by most surgeons. This operation has disadvantages, and in many cases I believe is positively contraindicated; an extensive wound is made which opens the space between the rectum and the posterior surface of

the prostate, a proceeding unnecessary, even when the suppuration has extended outside of the capsule. When an abscess is small it is sometimes hard to find by this method. The operation unless performed early does not prevent the opening of the abscess into the urethra and the formation of a urinary fistula. If the abscess has spontaneously opened into the urethra before the operation, the danger of fistula is increased. Multiple abscesses are difficult to manage by this operation. In cases of periprostatic abscess, the anterior wall of the rectum may be seriously weakened by the disease, and there is then danger of a rectal fistula if the prerectal incision is used. It has seemed to me that the convalescence after this operation is unnecessarily prolonged, and that it does not meet the necessities of these cases as efficiently as a median perineal incision.

I treat all forms of prostatic abscess by opening them into the prostatic urethra through a median perineal incision, whether the pus is confined within the capsule or has extended outside the capsule, whether it is above the triangular ligament or has extended into the perineum or into the ischiorectal fossa. I am convinced that this operation is sound in principal, and that abscesses thus treated heal promptly and with less danger to the patient than by any other method. I am sure that this operation will commend itself to those who will properly perform it and take the trouble to carry out the treatment in detail.

The technique of the operation is simple. The patient is placed in the lateral lithotomy position; the membranous urethra is opened upon a staff; the prostatic urethra is dilated and explored by the finger; a finger of the other hand is passed into the rectum, and between the two fingers the extent of the abscess cavity can be defined. It is easy to appreciate by the sense of touch the part of the prostatic urethra over the abscess cavity; and the openings when the abscess has discharged spontaneously into the urethra can be felt easily. The abscess is opened by tearing with the finger through the mucous membrane; its cavity is explored, any fibrous bands which traverse it are broken down, its floor is made level with the floor of the urethra, and the opening in the urethral wall is enlarged sufficiently to insure thorough drainage. This is done entirely with the finger; to do it efficiently requires experience; rough and unskilful manipulation may cause severe hemorrhage. A catheter, No. 28 F or No. 30 F, is introduced through the perineal wound into the bladder, and is retained by tapes fastened to a waistband. One or

two strips of gauze are passed alongside of the tube to the edge of the abscess cavity, but the latter is not packed. The tube and gauze I remove on the third day, sometimes on the second. The case is then treated as a perineal section. The patient passes all the urine through the urethra before the end of the second week; the perineal wound is healed at the end of the third or fourth week. Patients are out of bed at the end of the first week. Some cases require treatment of the posterior urethra for a short time after the complete closure of the perineal wound.

In cases of periprostatic abscess in which the pus is above the triangular ligament, the abscess cavity is opened and explored by the finger through the perineal incision: this is possible provided the floor of the membranous urethra is thoroughly cut to the apex of the prostate so as to divide the lower border of the triangular ligament. A large abscess between the rectum and the prostate is to be drained by a strip of gauze passed into the cavity alongside of the perineal tube; this is removed at the end of twenty-four or forty-eight hours. When the abscess has extended into the ischiorectal fossa an additional incision is necessary to drain this space. Perineal abscess beginning in or about Littré's or Cowper's glands or the intrabulbar glands, should be similarly treated. The treatment of these cases I have considered elsewhere.*

The treatment after operation is an important element in the quick and thorough recovery of these cases. It requires time and personal attention, but the result is an ample recompense for the trouble. I append a few cases illustrative of the points I have endeavored to make clear in this communication. These are selected as fair examples from a great many similar cases treated in my service at Bellevue Hospital.

Case I.—*Prostatic Abscess with Right Epididymitis, almost Complete Retention of urine; Symptoms temporarily relieved by Spontaneous Opening of the Abscess into the Urethra; Recurrence of Symptoms; Operation; Recovery.*—Timothy N., aged 29 years; United States; single; admitted April 7, 1902. First gonorrhœa when 27 years of age; discharge lasted five months; was treated by injections. Three weeks before admission he had a second attack of gonorrhœa. On April 1 he developed an epididymitis, which confined him to bed for a week; for this he was admitted to the hospital.

* Observations upon the cause and treatment of perineal abscess and of periurethral suppurations above the triangular ligament. Medical Record, New York, October 28, 1905.

On admission he had a right epididymitis and posterior urethritis. Two days later he began to have frequent urination with pain and rectal tenesmus and almost complete retention of urine.

Rectal touch showed the prostate to be enlarged, especially on the left side. It was hot and throbbing, no fluctuation. He was treated by hot applications to the perineum, and the pain diminished, but the swelling in the prostate increased in size, and on April 12, there was fluctuation near the apex of the gland. The prostate seemed to be smaller. There was a profuse discharge from the urethra, the abscess having opened spontaneously. On April 13 the amount of discharge was increased, pain in perineum and the rectum had greatly lessened. Patient was passing urine freely.

April 14 the discharge was still profuse.

April 15 prostate was again swollen, but a marked depression could be felt on the right side, which when pressed upon caused profuse discharge of pus from the urethra.

On April 19 operation was performed. On opening the membranous urethra the perineum, introducing the finger into the prostatic urethra, several small openings on the right side of the urethra were felt leading into an abscess cavity. At this point an opening was made with the finger and the abscess drained.

On April 22, the drainage and the bladder tube were removed. May 3 patient passed all urine through the urethra and made a satisfactory recovery.

Case II.—*Prostatic Abscess with Complete Retention of Urine, requiring the Use of Catheter for Ten Days; Symptoms relieved by the Spontaneous Rupture of the Abscess into the Urethra; Operation Refused; Persistent Urethral Discharge.*—James P., aged 44 years; widower; England; admitted March 22, 1902. Had gonorrhœa when 24 years of age; the discharge lasted two months; no complications. Five weeks before admission he contracted a second gonorrhœa; was treated by internal medication; no local treatment. Three days before admission he began to have frequent urination, severe burning sensation along the urethra with rectal tenesmus and pain in the perineum. These symptoms increased, and on the day before admission he had complete retention of urine, which persisted until his admission to the hospital.

Examination showed a slight purulent discharge from the urethra, which contained gonococci.

Rectal touch showed the prostate swollen and indurated, especially the right lateral lobe. There was œdema of the anterior rectal wall. As the result of straining at urination, he had a number of inflamed hæmorrhoids. The bladder was distended above the pubes. The urethra was free of stricture. Temperature was 100° F.; on the 30th it rose to 104° F., and then fell to normal on the 2d. A No. 14 F catheter passed easily and without great pain and forty ounces of urine were withdrawn; there was marked atony of the bladder. The urine was cloudy with pus, acid, contained albumen, but no casts. The catheter was passed at regular intervals, and the bladder was washed out with sterile saline solution once a day. On April 6 he was able to pass a few ounces of urine, which

contained about one-third pus by bulk. The prostate was smaller; the abscess had ruptured and was draining into the urethra. Catheterism was continued until April 9, after which the patient was able to empty his bladder completely. The pus in the urine continued to diminish in quantity, but never entirely disappeared. On April 18 the patient was discharged at his own request. The prostate then was nearly normal in size; he was passing water about five times during the day and three times at night. Urine contained about 2 per cent. of albumen, excess of urates, moderate quantity of pus, and some red blood-cells. Operation was refused by the patient.

Case III.—*Prostatic Abscess; Perineal Abscess; Double Epididymitis; Urethral Stricture; Abscess opened; Relapse of a Year Later; Slight Urine Leakage; Operation; Cure.*—J. M., aged 31 years; United States; single; admitted December 13, 1901. He had had a number of attacks of urethritis, the last attack was one year before admission. Two weeks after the urethral discharge appeared he developed an epididymitis upon both sides. An abscess then formed in the perineum, which was opened by simple incision at Fordham Hospital, New York. The wound healed promptly. A week before admission the urethral discharge returned, and two days before his admission a swelling in the perineum appeared at the seat of the former abscess; this increased rapidly in size and became very painful. The urine contained pus. There was a swelling in the perineum beneath the deep layer of the superficial fascia situated to the right of the median line; it was red and painful upon pressure.

Operation, December 14. Median perineal urethrotomy. A narrow stricture was found at the bulbomembranous junction, which was cut internally to No. 30 F. The abscess cavity contained about an ounce of pus, very little if any urine. It communicated by a small opening with the urethra behind the bulb. A small abscess found on right side of membranous urethra which had opened into the urethra; another abscess in the right lobe of the prostate. These were drained into the urethra. The bladder tube was removed the second day. Discharged cured January 11, 1902.

Case IV.—*Prostatic Abscess with almost Complete Retention of Urine; Operation; Recovery*—William J. C., aged 32 years; Irish; single; admitted March 29, 1902. Fourteen days before admission and three days after exposure he noticed a discharge from urethra. On the third day of the disease he began to have difficulty in passing water, with great frequency, pain and tenesmus. He applied at a dispensary, where he was treated by injections and by massage of the prostate. He had repeated chilly sensations and he thinks some fever. He had never had complete retention, but was obliged to pass water from fifteen to twenty times a day with much pain. Complained of a dull, heavy, aching sensation in the perineum. He had little apparent urethral discharge, the urine was dark colored. Specific gravity, 1027, contained albumen, large amount of pus, excess of urates.

Rectal touch showed a large, hard, indurated right lateral lobe of the prostate without fluctuation, tender to pressure. Temperature on admission was 99° F. On the following day it rose to 101° F. A small point

of fluctuation then was detected near the apex of the prostate. The symptoms continuing, operation was performed April 1. An abscess was found on the right side of the urethra which had not opened into the canal. This was opened and drained into the urethra. Patient was discharged cured on April 16.

Case V.—*Prostatic Abscess opening spontaneously into the Urethra; Retention of Urine persisting, the Abscess was drained by Operation; Cure*—James C., aged 29 years; United States; single, admitted January 27, 1902. Two weeks before admission he had first attack of gonorrhœa; no treatment. On the fifth day he began to have frequent urination, burning and stinging pain in the urethra and perineum. He applied at a dispensary; and was treated by internal medication. Day before admission he had complete retention of urine and increased pain in perineum. On admission he was suffering severely from retention of urine. His temperature was 99.5° F.

Rectal touch showed the prostate enormously swollen, especially on the right side. During this examination an abscess was ruptured into the urethra, causing the discharge of a large quantity of pus. A soft catheter passed easily; thirty-two ounces of urine were withdrawn; it was very cloudy, dense white sediment, acid, large percentage of albumen, no casts. As the retention persisted, operation was performed on January 29, 1902. Exploring the prostatic urethra, a large cavity opening into the canal was found on the right side. The opening was small in size and was near the apex; this was enlarged by the finger. The bladder was drained.

January 31 all drainage was removed.

On February 4 patient developed an epididymitis on the left side, which confined him to bed for about a week. He was discharged on February 25 cured.

Case VI.—*Relapsing Prostatic Abscess; Retention of Urine; Abscess opening into Urethra during Catheterism; Imperfect Drainage requiring Operation; Recovery*—C. M., aged 23 years; United States; single; admitted February 6, 1904. Four months before had gonorrhœa; for this he was treated by injections. Three weeks before admission symptoms of pain and frequent urination began, resulting in complete retention of urine; he was relieved by catheterism. Two days later he again had retention of urine and was relieved by catheterism. Since then he had had retention of urine twice; relieved by catheterism. Following the passage of the catheter a week before admission, a large quantity of pus was discharged from the urethra.

On admission he had a urethral discharge; urine was cloudy with pus; he had left epididymitis; the prostate was swollen, especially the left side.

Operation on February 7, 1904. Perineal section. The finger found several openings in the mucous membrance of the prostatic urethra which led into abscess cavities; these openings were united by the finger and the abscess drained into the urethra; a hooded perineal tube was inserted, as the hæmorrhage was quite profuse and the perineal wound was packed with gauze.

On February 10 the tube was removed, and for several days there was almost complete vesical incontinence.

On February 17 he passed some urine through urethra.

On February 26 he had perfect control of his urine and passed all of it through the urethra. He was discharged March 15 perfectly well.

Note.—One year afterwards this patient was seen. Urine clear; he had perfect control; No. 26 F sound passed easily. Prostate by rectal palpation normal. He had contracted syphilis, and is now under treatment for this malady.

Case VII.—*Tuberculous (?) Prostatic Abscess; Pericowperitis; Perineal Abscess; Incomplete Operation; Recurrence of Abscess requiring Second Operation; Slow Convalescence; Perineal Fistula*—F. S., aged 17 years; single; admitted October 10, 1900. Denies all venereal disease. His present illness began September 1. He had painful urination with pain before and after the act. Seven or eight days later he began to have pain in the perineum, and a small swelling appeared; this had grown progressively larger. On September 15 the frequency of urination increased, and he had partial retention of urine. On admission his temperature was 99° F.

Examination showed in the perineum a median oblong swelling over which the skin was reddened. Distinct fluctuation. The urethra was free from stricture. There was a urethral discharge, which did not contain gonococci, but contained a rod-shaped bacillus resembling the tubercle bacillus.

Operation on October 12. Median perineal section: an abscess was found beneath the deep layer of the superficial fascia. This was opened and drained. Membranous urethra was opened. Examination of prostatic urethra failed to detect any sign of abscess. The tube was removed on second day. Patient did well until the eleventh day after the operation, when an abscess was discovered at the bottom of the perineal wound at the side of the urethra extending towards the groin. This was opened and drained, and on the 25th another abscess was discovered in the ischiorectal fossa. A second operation was performed on October 26. Membranous urethra was reopened; the finger introduced into the prostatic urethra found a large abscess in the left lobe, which communicated with that in the perineum, and there was also suppuration about Cowper's glands. These abscesses were opened into the urethra. An oblique incision was made beginning at the upper margin of the perineal opening and extending towards the left groin, which opened an abscess cavity in this situation; the abscess in the ischiorectal fossa was opened by a separate incision. The abscess cavities were drained and a tube was placed in the bladder. The patient's general condition was unsatisfactory and the wounds were slow in healing. He was discharged on October 29 with a small perineal sinus which had not healed, and through which a few drops of urine escaped at each urination.

Case VIII.—*Prostatic Abscess and Perineal Abscess; almost Complete Retention of Urine; Abscess opened spontaneously into the Urethra with Inefficient Drainage; Operation; Recovery*—J. O'R., aged 25 years; married; admitted December 6, 1900. He contracted a gonorrhœa in 1894,

which lasted for six weeks, and again had a gonorrhœa in 1898, and had had a relapsing urethral discharge ever since. About three weeks before admission he began to have pain in the perineum, which was increased by exercise and on defecation. He had same difficulty in passing water, but never had complete retention of urine. Three or four days later a swelling appeared in the perineum. This gradually increased in size. He had chilly sensations with fever. On admission, temperature was 100° F. Urine was foul, contained large quantity of pus, many large shreds, albumen; specific gravity, 1018. There was in the perineum an ovoid swelling, more marked on the right side. Skin over this was red. Tumor was not very well defined, edges were hard. There was distinct fluctuation. There was no stricture. Right side of the prostate was enlarged and tender. Perineal operation, December 9, 1900. On cutting through the deep layer of the superficial fascia, an abscess was opened containing about eight ounces of pus. Membranous urethra was opened, and a large abscess cavity was found by the finger on the right side of the prostate communicating with the urethra; this was drained. Tube was put in the bladder. Tube and drainage were removed on fourth day. Patient passed his urine through the urethra on the ninth day and made a satisfactory recovery.